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THE DEVELOPMENT OF OUR KNOWLEDGE OF ANIMAL TRYPANOSOMIASES*

By

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INTRODUCTION

I had intended to address you on "Recent Advances in Our Knowledge of Animal Trypanosomiases," but my definition of "recent" with its assumption that my hearers were all acquainted with earlier work, might not have been acceptable. It seemed safer, then, that I should sketch very briefly the development of our knowledge of these diseases from the very beginning, dwelling on nothing, of necessity omitting many names and occasions that different members of my audience will think I should at least have mentioned, yet endeavouring to touch on every aspect so that as any point of particular interest arises it may be noted and serve subsequently to promote a question or discussion.

To go straight from the beginning of a long history to its end without a break is exhausting, so entirely for convenience I have chosen the year of the outbreak of the first world war as an arbitrary date at which to draw breath before plunging into a second period that takes us to the present time.

DEFINITION

TRYPANOSOMIASES may be defined as a group of several allied diseases, each of which is due to infection with a specific trypanosome.

First Period: Before 1914

Although trypanosomes are protozoa, yet they are easily seen under the microscope, and it is probable that all the important pathogenic species have been isolated and studied. In consequence, the various trypanosomiases can for most practical purposes be separated from each other. But this separation is not in all cases definite, for there is still division of opinion regarding the specific status of some of the causal organisms.

J. Huxley, in his book "Evolution," considers a quite reasonable definition of the term species is that given some years ago by Dr. Tate Regan when Director of the Natural History Museum at South Kensington—namely, that a species "is a community or a number of related communities, whose distinctive morphological characters are, in the opinion of a competent systematist, sufficiently definite to entitle it, or them, to a specific name." Huxley then points out that the difficulty with this definition lies in the term *competent*, and experience teaches us that even competent systematists do not always agree as to the delimitation of species. To this difficulty may be added another due to the small size of unicellular organisms: the fact that two trypanosomes, apparently morphologically identical, may yet possess biological characters, notably pathogenicity, which, in the opinion of some competent systematists, are constantly and therefore specifically different, while other systematists consider that if trypanosomes are morphologically identical they must be considered one species, divided into biological races by their differences in host relations.

While there is thus still an overlapping of trypanosomiases due to our inability to separate with certainty all their causal organisms, we can go much farther than those who knew the diseases before trypanosomes were discovered. In those days the overlapping of syndromes led of necessity to the lumping into groups of a number of trypanosomiases that are now recognised as separate diseases. These groups were given common names that still persist; for example, in Africa the two trypanosomiases of man are both known as sleeping sickness, while several trypanosomiases of domestic animals retain the common title of nagana, a corruption of the Zulu word "ngana," meaning feeble or weak—the state to which the diseases reduce affected animals.

Long before modern times the more outstanding trypanosomiases or groups of trypanosomiases were well-known diseases. Stock-owning tribes of many parts of Africa have not only been familiar with nagana from time immemorial; they have also recognised the fact that it was given to animals by the bites of tsetse flies. The word "tsetse," according to Fuller, is a Selika term introduced into the English language by the great hunter, Gordon Cumming, in 1850. About the same time another hunter, Vardon, when in the valley of the Limpopo River, caught a tsetse on his horse, and took it to England, where Westwood gave it the name *Glossina morsitans*, and for many years it was thought that all tsetses were of this one species.

There are historians who go so far as to associate the fourth plague of Egypt—"There came a grievous swarm of flies" (*Exodus* viii, 24)—with the fifth—"and all the cattle of Egypt died" (*Exodus* ix, 6)—and think that this legend is based on still older ones of great stock losses, following what to-day would be termed tsetse-fly encroachment. However, what is probably the first scientific description of nagana is that given by Livingstone in his "Missionary Travels," etc., published in 1857.

Far from lands where tsetses occur, diseases now known to be trypanosomiases have for long been recognised as animal scourges, *e.g.*, surra, notably in India; mal de Caderas, in South America; and murrina, in Central America. Also, according to Leclainche, the trypanosomiasis now known as dourine was well known to the Arabs in the eleventh century, though definite records of this disease date only from 1796, when Ammon observed it among the horses of the Prussian stud at Trakehnen.

But the recognition of trypanosomiases *as such* began with the discovery of the first pathogenic trypanosome, the cause of surra, by Evans in 1880. Although at first it was named *Spirochaeta evansi* by Steel in 1885, yet the next year Crookshank recognised its true nature and the name was changed to *Trypanosoma evansi* (Steel, 1885). It should be noted that,

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in accordance with Article 23 of the International Rules of Zoological Nomenclature, although the species was transferred from the genus *Spirochaeta* to that of *Trypanosoma*, the name of the author of the specific name is retained but placed in parentheses.

The next great advance dates from 1894, when David Bruce went to Zululand at the request of the South African Government to study nagana. There, his masterly researches revealed the trypanosomal nature of the scourge, and he showed by experiments that the parasite was transmitted by tsetse flies. He also established the fact that wild game act as a reservoir of the trypanosome. In 1896 Bruce sent to England an infected dog, and the strain of trypanosomes thus obtained was the source of material for innumerable researches in many European laboratories. Among the first to study it were Plimmer and Bradford, who, in 1899, named it *Trypanosoma brucei* in honour of its discoverer.

By strange chance this strain of *T. brucei* was an unusual one, described correctly by Plimmer and Bradford as monomorphic, whereas the most obvious characteristic of typical *T. brucei* as known to-day is its polymorphism. We know now that ordinarily *T. brucei* must be considered a polymorphic trypanosome of high virulence for dogs and some other animals, yet occasionally a monomorphic strain of low virulence is encountered, and it was such a strain that was first sent home; in fact, a dog infected with an ordinary strain of *T. brucei* would not have reached England alive. The occurrence is strange because such monomorphic strains are rarely derived directly from animals naturally infected with nagana; they usually develop from normal polymorphic strains after these have been passed directly by syringe through a long series of vertebrate hosts. At any rate, this description of *T. brucei* as a monomorphic organism created many difficulties. The resemblance of this newly discovered parasite to *T. evansi*, the cause of surra, and the only other trypanosome known at that time to be pathogenic, was so obvious that many people thought the two parasites should be considered one species. Until it was realised widely that the *T. brucei* sent to England was atypical it was natural for discoverers of polymorphic trypanosomes in various parts of Africa to decide that whatever the relation of these parasites to one another, they were certainly not *T. brucei*. And so arose some of the many names now considered to be synonyms of *T. brucei*. It was natural, too, that at that time Bruce should not suspect nagana to be a group of diseases transmitted by many species of *Glossina*: he thought of it as a single disease due to a specific trypanosome, *T. brucei*, transmitted by a specific biting-fly, *G. morsitans*.

The first decade of the twentieth century saw many advances in knowledge of trypanosomiases. One of the earliest, yet most important, was the finding by Ford of a parasite in the blood of a man in the Gambia that was recognised as a trypanosome and given the name *Trypanosoma gambiense* Dutton, 1902. The following year Castellani found a trypanosome in the cerebro-spinal fluid of a case of sleeping sickness in Uganda—a trypanosome soon shown to be the same as that seen by Ford in human blood—and these discoveries formed a good basis for the very extensive studies of the "gambiense" form of sleeping sickness that have been going on ever since.

Meanwhile the year 1901 saw the naming of two new species of trypanosomes pathogenic to domestic animals: Doflein gave the name *Trypanosoma equiperdum* to a parasite isolated from a case of dourine and recognised as the cause of the disease, while Voges gave the name *Trypanosoma equinum* to the parasite similarly implicated in the South American disease called mal de Caderas.

In 1902 *Trypanosoma theileri* Laveran received its name, though at the time it was erroneously believed to be the cause of what is now known to be anaplasmosis. In 1904 Broden discovered the important species *Trypanosoma congolense* in sheep and a donkey in the Congo, while in 1905 the almost equally important *Trypanosoma vivax* Ziemann received a name indicative of its great motility.

The next scene in this chronicle of discoveries of pathogenic trypanosomes is Brazil, where Chagas accomplished a remarkably comprehensive piece of research. As related by Wenyon: "*Trypanosoma cruzi* was first discovered by Chagas in 1907, and two years later described by him as a parasite of the reduviid bug, *Triatoma megista*. The bugs were known to attack man in certain parts of Brazil, and Chagas discovered crithidia forms of a flagellate in the hind-gut of specimens of the bug collected at Minas. Some of these were allowed to feed on a marmoset, *Hapale penicillata*, which three weeks later showed trypanosomes in its blood. The trypanosomes were found to be inoculable to dogs, guinea-pigs and rabbits. Extending his observations, Chagas ultimately found the organism in a cat, and later in children who suffered from a wasting disease which had long been known in the country. Chagas first placed the trypanosome in the genus *Trypanosoma*, but later, on account of its peculiar intracellular mode of development as leishmania forms, created the new genus, *Schizotrypanum*, for its reception. This name was chosen because it was believed that reproduction took place by schizogony, but it is now known that multiplication, though occurring within cells in the leishmania stage, is by the usual method of binary fission, so that it is preferable to retain the trypanosome in the genus *Trypanosoma*."

After Chagas' classical article was published in 1909, only three more years were to elapse before the tale of discoveries of pathogenic trypanosomes was completed: in each of the three years one new species of trypanosome was named. All three are African. The first, *Trypanosoma rhodesiense* Stephens and Fanthem, 1910, the cause of all African sleeping sickness not due to *T. gambiense*, has been the subject of much research on account of the very great importance of the disease it causes, and it has also been the subject of much controversy regarding its relationship to the two other polymorphic trypanosomes, *T. gambiense* and *T. brucei*. It is particularly its relationship to *T. brucei* that it is so interesting, for in every way the two trypanosomes appear to be identical except that the one is highly pathogenic to man, and the other is harmless. Unless, or until, this vital difference is broken down by either (*a*) a strain of undoubted *T. brucei* being rendered pathogenic to man, or (*b*) a strain of undoubted *T. rhodesiense* being rendered permanently avirulent to man while retaining all its other properties, one must consider the two species as separate.

Both the remaining trypanosomes were discovered and named by Bruce and co-workers. The first, *Trypanosoma uniforme* Bruce, Hamerton, Bateman and Mackie, 1911, was first studied in Uganda, and it, also, has required champions, notably Hoare, to enable it to retain its claim to be a valid species, since it differs from *T. vivax* only by being smaller. The other, *Trypanosoma simiae* Bruce, Harvey, Hamerton, Davey & Lady Bruce, 1912, first found in Nyasaland, belongs to the same group of trypanosomes as *T. congolense*, but can assume a degree of virulence towards pigs, monkeys and camels that is never attained by any other species.

Two other trypanosomes are found in domestic animals without provoking disease. They are *Trypanosoma melophagium* Flu, 1908, of the sheep, transmitted by the ked *Melophagus ovinus*, and *Trypanosoma theodori* Hoare, 1931, of the goat, transmitted by the hippoboscid fly, *Lipotena caprina*.

To-day, when the tendency is towards still further reduction of the dozen or so species of pathogenic trypanosomes that most workers consider as truly independent, it is difficult for a young worker entering the field of trypanosomiasis research to realise the confusion of names that existed up to and even after the first world war. In order that he may realise something of what he is spared he might glance through some of the literature of those earlier days: read, for example, such an article as one by Braun and Teichmann (1914) on "Experiences connected with the Animal Trypanosomiases of German East Africa." The translation of the opening paragraph of a section entitled "Paranagana" is as follows: "Among the African trypanosomes there have been described a whole series characterised by their small size and the shortness of their flagellum. They have been given different names (*Trypanosoma congolense* Broden; *T. pecorum* Bruce; *T. nanum* Laveran; *T. Frobeniusi* Weissenborn). Bruce has endeavoured to bring these trypanosomes into one group, to which he has given the name *T. pecorum*, and with which he considers *T. dimorphon* should also be included." It is only within very recent years that the sorting out of trypanosomes has been brought to the stage that allows observations and experiments wherever made to be of full value to all other workers because there is no doubt to which of the following species reference is made:

T. theileri Laveran, 1902
T. brucei Plimmer and Bradford, 1899
T. gambiense Dutton, 1902
T. rhodesiense Stephens and Fantham, 1910
T. congolense Broden, 1904
T. simiae Bruce *et al.*, 1912
T. vivax Ziemann, 1905
T. uniforme Bruce *et al.*, 1911
T. evansi (Steel, 1885)
T. equinum Voges, 1901
T. equiperdum Doflein, 1901
T. cruzi Chagas, 1909.

Concurrently with the discovery of new species of pathogenic trypanosomes, advances were being made in the ways these parasites are transmitted. It has been recounted how from time immemorial it was common knowledge that the bites of tsetses could convey nagana, and that it was Bruce, in Zululand, who showed that *T. brucei* is transmitted by *G. morsitans*. A few years later, in 1903, the same investigator, working with Nabarro and Greig in Uganda, showed that the trypanosome of sleeping sickness, *T. gambiense*, was similarly transmitted by *G. palpalis*, and shortly afterwards other species of tsetse became strongly suspected of being able to act as similar vectors.

But until 1909 all transmission experiments seem to have been of the nature of a mechanical transmission, that is by flies which start a meal on an infected animal, and complete it, albeit after some hours, on a clean susceptible one. In that year Kleine, working in German East Africa, fed *G. palpalis* for three successive days on two sheep and a mule infected with *T. brucei*. From the fourth day onwards they were fed on a clean fresh sheep or ox every day. The animal used on the 18th day became infected, while those fed upon earlier remained healthy. Thus cyclical development in the tsetse was first demonstrated, and within a few years much was learnt of the process, including the fact that this is not uniform for all species; so most attempts to classify trypanosomes take into account the different modes of development within tsetse flies.

In nature, transmission of trypanosomes may be either cyclical or mechanical. Where domestic stock are associated with tsetse flies, infection is usually cyclical, but all forms of trypanosomiases can be spread by interrupted feeds of biting flies, including tsetses, and this is the normal method of spread of some, *e.g.*, surra. The human disease due to *T. cruzi* is normally transmitted cyclically by its invertebrate host, a reduviid bug, and the equine trypanosomiasis, dourine, mechanically by coitus.

The developmental forms in the insect vector resemble cultural forms. It was in 1903 that McNeal and Novy first succeeded in cultivating trypanosomes—first *T. lewisi* of the rat and, later, *T. brucei*.

From the earliest days of scientific investigation of trypanosomiases it was recognised that wild animals of many species were not only reservoirs from which tsetses often draw their infection, but that their blood was the flies' main food, and ever since there have been steady additions to knowledge of the respective importance of different species of wild hosts in these respects. To-day it is recognised that blood is not merely the main food of tsetses: it is their sole food.

By 1914 a great deal was known about the distribution and aetiology of the various trypanosomiases, and many measures to combat these diseases were in force; for example, by wholesale movement of natives from particularly bad areas of sleeping sickness; by clearing major vegetation from the neighbourhood of villages and watering-places, to render these unfavourable to tsetses, and by encouraging the destruction of game, particularly buffalo.

But medicinal treatment was poorly developed in 1914. The first trypanocidal drug to be discovered was trypan red, found by Ehrlich and Shiga, in 1904, to cure mice infected with mal de Caderas. In 1906, Mesnil and Nicolle showed that the related drug, trypan blue, was more valuable, and in the same year they also obtained promising results with a variant of trypan blue, Afridol violet—of especial interest because it was from this that the enormously important drug Bayer 205 ultimately was developed. But this development was in the future. In 1914, arseno-phenylglycin, soamin and atoxyl were the medicaments which gave qualified success in the treatment of "gambiense" disease, but no success in "rhodesiense" sleeping sickness, which was incurable. Treatment of animal trypanosomiases was likewise very unsatisfactory. Holmes had worked out an elaborate 21-day treatment of surra, using a combination of arsenious acid and atoxyl; and many other drugs, alone or in combination, were bring tried for this and the various African diseases. Only one held out real promise, namely, tartar emetic, and although veterinarians were still only feeling their way towards its correct usage, there already existed numerous records of cures attributed to it. Veterinarians in the tropics will agree with the statement of Manson-Bahr that "the modern era of the scientific application of antimony commences with the employment of intravenous injections of tartar emetic as a potent remedy in tropical diseases, and this stage will be regarded by future generations as one of the most important milestones in the history of therapeutics." The trypanocidal action of arsenic was already known in 1906, when Nicolle and Mesnil suggested that on account of the chemical relationship of the metals, antimony compounds should be tried in the treatment of trypanosomiases. The proposal was carried into successful practice by Plimmer and Thomson, who, in 1907, gave injections of both sodium and potassium antimony tartrate subcutaneously to rats experimentally infected with trypanosomes and noted that the drugs had an extraordinarily significant and remarkable action on the circulating trypanosomes. About the same time, Mesnil and Brimont also arrived at the conclusion that tartar emetic had strong

trypanocidal action, and is easily supported when given intravenously though not when given subcutaneously. The results of these experiments on laboratory animals were seen translated into practical attempts to cure naturally contracted cases of human and animal trypanosomiases with the aid of tartar emetic as principal agent. By 1914 this drug had already proved disappointing in its action on the human diseases, and results with domestic animals were erratic and, on the whole, unsatisfactory.

Second Period: After 1914

Distribution.—Two events are of outstanding historical interest: one represents enlargement of the areas where trypanosomiases were known to be problems; the other a notable contraction. In 1919, Leger and Vienne discovered a trypanosome in cattle in Venezuela, and the view is now accepted that the parasite is *T. vivax*, until then considered an exclusively African species occurring only where tsetses were also found. Since then the same parasite has been found in many other parts of the New World. In 1920, Watson announced that dourine had been stamped out in Canada as the result of a slaughter policy based on diagnosis by the complement fixation test applied to 40,000 horses between 1912 and 1919.

Aetiology.—Since 1914 the main work in connection with the pathogenic trypanosomes themselves has been a consolidation of knowledge rather than spectacular new discoveries. Methods of reproduction have been discussed frequently without *definite* advances in knowledge being made. Trypanosomes divide by longitudinal fission, and all gross stages of this process are observable in both fresh and stained preparations, but details of nuclear division are naturally obscured by the small size of the organisms. Therefore, whether this division is mitotic or amitotic or something between the two is not easy to observe; it is difficult either to confirm or refute a statement regarding chromosome numbers. But if there is nothing improbable about claims that mitosis can be demonstrated, I do not think the same can be said of claims regarding syngamy. I still hold the opinions expressed by Wenyon on page 340 of his famous text-book. This author maintained that all so-called conjugating forms of pathogenic trypanosomes could be explained as dividing individuals or as the casual association of two flagellates. He concluded: "It is clearly evident that, of the numerous statements which have been made regarding the occurrence of sexual differentiation and syngamy among the Trypanosomidae, not one had any evidence to support it." My opinions have not been changed by the recent bold statement by Fairbairn & Culwick that "syngamy has been found to take place in *T. rhodesiense* and *T. simiae*"; their claim must first be confirmed by others. Regarding cultivation, recent years have seen a series of articles, commencing with one by Biocca in 1938, on the ability of pathogenic trypanosomes to multiply in chick embryos. It was just before 1914 that Oehler showed that infection could be set up by the inoculation of a single trypanosome.

Diagnosis.—A simple test for the recognition of all but the earliest cases of surra in camels is the formol-gel test (or Gaté-Papacostos reaction) as applied by Plantureux in 1923. In 1928 Bennet and Kenny stated that a better test was with mercury perchloride in place of formalin. The success of the test depends on a peculiarity of camel blood; it does not work with trypanosomiases of other domestic animals. With its help, and by treating reactors with B 205, Bennet has been most successful in controlling surra in the Anglo-Egyptian Sudan.

Transmission and Reservoirs.—Many valuable additions to knowledge have been made, but most are of the same nature as discoveries made previously and do not rank as historical events. However, special mention might be made of the long series of experimental transmissions of surra by many species of biting flies made by Nieschulz in Java and Sumatra. He used more than 14,000 insects of various kinds, of which tabanids proved to be by far the most important transmitters. In connection with reservoirs, probably the most important work of recent years is the long series of observations still being continued at Tinde, Tanganyika Territory; at first by Corson, later by Burtt & Fairbairn, and now by Fairbairn & Culwick. The main object of this painstaking research is to find out for how long game and domestic animals can continue to maintain *T. rhodesiense* in an unchanged state of pathogenicity to man. For more than 12 years a strain of this trypanosome derived from a case of sleeping sickness has been passed from antelope to antelope and from sheep to sheep by cyclically infected *G. morsitans*, without loss of virulence to man. Meanwhile, Burtt made the interesting discovery that the ability of tsetses to transmit trypanosomes is influenced by the environment to which they are submitted as pupae.

Treatment.—It is in the field of chemotherapy that advances in knowledge have been most spectacular. At Tinde the experiments just referred to necessitate the use of human volunteers for testing periodically the pathogenicity to man of the *T. rhodesiense* maintained there. In 1914 such tests would have been out of the question, though it is interesting to recall that as long ago as 1913 Taute inoculated himself with *T. brucei* to demonstrate this was different from *T. rhodesiense*, at that time the cause of incurable disease of man. A few years later, thanks to the discovery of Bayer 205, "rhodesiense" sleeping sickness in its early stages could be cured with such certainty that hundreds of native volunteers have submitted cheerfully and almost without risk to inoculation with *T. rhodesiense* as an easy way of earning money.

The drug Bayer 205 was, according to Wenyon, first introduced in Germany in 1920 by Haendel & Joetten and by Mayer & Zeiss. It has remarkable curative and prophylactic properties. As stated above, it revolutionised the prognosis in all early cases of "rhodesiense" sleeping sickness. In trypanosomiases of veterinary importance it is not so valuable, as equines are strangely susceptible to its toxic action; nevertheless, as early as 1922 Baermann used it curatively in surra, and he was also the first to describe its prophylactic action.

Another drug of value in sleeping sickness that first came into prominence about 1921 is tryparsamide, but this has not been found to be useful in animal trypanosomiases.

Developments in chemotherapy of animal trypanosomiases were at first mainly associated with greater experience in the use and of the limitations of tartar emetic. By 1919 the writer, who made much use of this drug during the military campaign in German East Africa, was able to state that he believed most cases of disease due to *T. vivax* and to *T. congolense* could be cured by a course of injections extending over a long period, and that sometimes a single injection sufficed to bring about this result. He found, however, that tartar emetic, like all other drugs which had been tried, was useless in the curative treatment of *T. brucei* infection of equines.

Improvement in antimony therapy was effected when the I. G. Farbenindustrie A. G. put antimosan on the market about 1929. Its trypanocidal action is the same as that of tartar emetic, but it is less toxic and it has the great advantage that it can be given subcutaneously. When the last war

started, a British substitute, stibophen, took the place of antimosan.

About 1933 the same German firm (who were also the discoverers of Bayer 205) produced yet another type of trypanocidal drug: Surfen C or Congosin, stated to be a quinoline derivative. From then until the last war drove German products off the open market, this drug was used fairly extensively, since a single intramuscular injection gave about as good results in infections due to *T. congolense* as a course of antimonials.

Another line of chemical attack on trypanosomiases started when Lourie & Yorke, in 1939, showed that several compounds of the aromatic diamidine series were strongly trypanocidal. Of these, pentamidine is of most interest to the veterinarian by reason of the long protection against trypanosomes of the *T. brucei* and *T. evansi* groups conferred by a single injection. Then came the phenanthridinium compounds first brought to public notice by Browning, Browning and Robb in 1940. The trypanocidal value of dimidium bromide and later members of this series of phenanthridinium compounds is almost miraculous, though unfortunately it is somewhat offset by the occasional severity of the local reaction when the drug is given subcutaneously or intramuscularly, and by the toxic action on the liver revealed spectacularly by occasional cases of photosensitisation. And now we have antrycide, of which we have heard so much recently. The tremendous value of this drug should not, however, make us neglect the continued study of the phenanthridinium compounds, for these also are of tremendous value at the present time.

Pathology and Pathogenicity.—Many scattered observations and experiments have been made all over the world in endeavours to find out how trypanosomes produce the various symptoms and, ultimately, cause death. No very clear picture has yet emerged, though it is now recognised that trypanosomes of the *T. congolense* group are distributed throughout the body in a different manner and behave differently from those of the *T. brucei* group. A notable contribution to the pathology of diseases due to the *T. brucei* group was made by an Italian, Peruzzi, attached temporarily to a League of Nations International Commission on Human Trypanosomiasis, working in Uganda in 1927.

Recent work by Fiennes, Jones & Laws suggests that the main lesions of *T. congolense* disease may be due to multiple infarction leading to parenchymatous degeneration and replacement fibrosis. Their cases, however, were of extraordinarily acute disease not representative of what usually takes place. The usual syndrome is of an anaemia that gives rise to no lesions which cannot be readily resolved once the causal organism is removed. Possibly of more significance is the work of French and some earlier workers on the increase in the globulin fraction and the proportionate decrease in the albumin fraction of the serum proteins that takes place in most, if not all, trypanosomiases.

Of interest in connection with pathogenicity is the revelation by Pellegrini only last year that *T. simiae* can cause as hyperacute disease in camels as it does in pigs and monkeys.

Immunology.—It is now generally recognised that immunity to trypanosomes acquired by normally susceptible hosts is of the kind usually called premunition; where the condition of resistance is accompanied by or even dependent on the survival of the parasite, so that an immunised domestic animal remains a reservoir of the trypanosome and a potential source of infection to other animals. For this reason research on immunisation finds qualified support, and all that has been done so far has failed to show any practical way in which domestic animals can be immunised reliably.

Epizootology and Control.—It is obvious when one considers the worldwide distribution of animal trypanosomiases that these diseases must not be thought of as being always associated with tsetse flies. Nevertheless, tsetse flies must remain indissolubly connected with trypanosomiases, and their elimination from any area is necessary before this can be used for stock-raising. Owing to the vast areas of Africa rendered inhospitable by these flies, special departments have been formed by Governments for the study and control of tsetses. The work of many investigators, among whom the late C. F. M. Swynnerton stands very high, has thrown much light on the bionomics of the score or so of species of tsetse flies, and it is now fairly clear that each of these species requires special measures for its eradication, and that there is no panacea. Four notable experiments on a very large scale have been tried in recent years: (1) In Zululand a trapping campaign was launched against *G. pallidipes*, and prosecuted almost regardless of cost; but in the end it failed. (2) In Tanganyika Territory, hundreds of square miles of fly-infested woodland of the kind that normally is subject to periodic veld-fires—which keep it open and suitable for both game and certain species of tsetses—were protected from fire for upwards of ten years, to see if the resulting densification of vegetation would render them unsuited to these flies. This great experiment, too, may be said to have failed to produce results of practical value. (3) In Southern Rhodesia a campaign launched against *G. morsitans* aimed at eliminating all dependable food supplies of the fly by systematic and controlled shooting of game. This experiment was successful, for not only was fly encroachment stopped, but thousands of square miles of infested country were reclaimed. (4) The endeavour, also in Zululand, to eliminate tsetses from a large area by means of spraying insecticides from the air. Final results are not yet to hand, but figures already published show sufficiently impressive results to justify the hope that full success may be achieved; at least when the area is a fly-belt well isolated from others.

Finally, my personal opinion is that the large tractor fitted with bull- or tree-dozer is the most important weapon for combating tsetses yet introduced into Africa. With the aid of a number of these powerful machines, supported by comparatively little hand labour, great areas of fly-infested woodland and bushland can be systematically turned into fly-free arable- or grass-land. This is being well demonstrated by the Overseas Food Corporation, whose clearing work to grow food-stuffs for Britain may at the same time prove to be the most important experiment in tsetse reclamation that has yet been made.

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Discussion

Owing to limited time the discussion that followed consisted mainly of a number of questions addressed to the lecturer, and his replies to these.

Mr. Peck pointed out that in British Somaliland surra was practically confined to camels. He asked if this were the case elsewhere in East Africa.

A. This is the case. In recent reports from the Anglo Egyptian Sudan, for example, *T. evansi* is always referred to as the cause of camel disease. Only in Mauritius are other animals reported to be frequently infected, but in this island there has been some confusion between *T. evansi* and *T. vivax*.

Mr. Newlands asked if the game of Mauritius harboured *T. evansi*.

A. This parasite is supposed to have been introduced into the island during the Boer war. It is not, therefore, truly indigenous. Some years ago it caused much mortality among the deer that have also been introduced, and pariah dogs died from eating the infected carcases.

Mr. Wilde asked if a careful comparison of strains of *T. vivax* derived from S. America with others obtained from Africa had been made. Also, was there any knowledge as to how *T. vivax* got to S. America?

A. Yes, careful comparison has been made, and it is surprising how closely the two kinds of strains resemble each other. It is a safe assumption that the parasite was taken to S. America in cattle which throughout hundreds of years have been carried in many ships from West African to East American ports.

Mr. Reid and Mr. Stuchbery both raised the question as to whether the wide use of trypanocidal drugs might cause the production of drug-resistant strains of the various parasites.

A. Undoubtedly there is a danger, which becomes greater in proportion to the extensiveness of the use of the drug. No doubt strains of drug-resistant trypanosomes are constantly being produced even to-day, but so long as there remain large numbers of untreated wild or domestic animals harbouring trypanosomes in the neighbourhood, these few drug-resistant strains are being as constantly swamped by super-infections. With the wider use of a particular drug, an effort should be made to avoid giving subcurative doses.

Mr. Beakbane asked to what drug-fastness is due.

A. It has been suggested that drug-fastness is possibly related to acquired impermeability of the trypanosome membrane to the drug. This acquisition arises as a mutation, and the great difference in the mutative power of even chemically related drugs has to be taken into consideration in assessing their value. For example, tartar emetic has an exceedingly low mutative power and atoxyl a very high one.

Mr. Newlands asked if tsetse flies were sterilised when they fed on animals impregnated with a trypanocidal drug.

A. I know of no such claim for any drug.

Mr. Newlands then asked if game fences were of definite value in controlling trypanosomiases.

A. They can be of definite value, as demonstrated in both Southern Rhodesia and Bechuanaland, but only if used in conjunction with shooting of game and control of stock movement in their neighbourhood.

Mr. Burns asked what should be done if sleeping sickness broke out on a stock-route. Would there not be a danger of the animals acquiring *T. rhodesiense* and subsequently infecting other flies, perhaps in a belt formerly free from this human infection?

A. Theoretically there is such a danger, though no concrete evidence of it having occurred has been published. As both pentamidine and Bayer 205 have excellent prophylactic properties, the use of one or the other before the animals entered the infected fly-belt might be considered. In French West Africa pentamidine is used as a prophylactic against *T. gambiense* in human beings, and an intramuscular injection of 4 or 5 mg. per kilo body weight is stated to give protection for six to eight months.

Mr. Aspinall asked if Mr. Hornby had heard of a recent claim that animals could be freed from *T. rhodesiense* by giving them a super-infection of *T. brucei*. The two parasites were said to conjugate and produce hybrids resembling the harmless (for man) *T. brucei* in their pathogenicity.

A. I have heard of this claim, and I look upon it as an interesting possibility. As I stated in my address, I am not convinced that syngamy of trypanosomes occurs, therefore I cannot at present believe any report of hybridisation. Of course, it is possible that *T. rhodesiense* may be eliminated from the body by a super-infection of *T. brucei* without this being the result of hybridisation.

Weekly Wisdom

". . . However great may be the energy locked up in a physical system, its effective utilisation is entirely dependent on the existence of differentials, whether of temperature, pressure or electrical potential. Without such differentiation the system will be inert and lifeless, and mobility can be imparted to it only by the application of external forces. Human beings are part of the physical universe, and while the physical analogy may not be perfect in its application to human society because other than physical influences come into play, it cannot be denied all relevance. Uniformity and incentives are incompatible. Visible inequality is a constant spur to ambition. A properly differentiated society may be self-regulating. Uniformity clamours for external control. . . ."—The Rt. Hon. Sir John Anderson, M.P. (*The Sunday Times*, June 19th, 1949).

THE DISTRIBUTION OF TUBERCULOUS LESIONS IN THE DOG AND CAT, WITH REFERENCE TO THE PATHOGENESIS*

By

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The cases presented here have been encountered during the course of routine general diagnosis and it is felt that an analysis of them may prove of interest, not only to the veterinary surgeon in general practice, but also to those concerned with public health. Tuberculosis of the dog and cat has received attention from various authors but there is no general agreement on the incidence and pathogenesis of the disease. The disease, in this area, appears to be more common than is generally supposed and it is possible that this may apply to other areas. No special search has been made for these cases of tuberculosis, and their relative frequency, together with the fact that many of them were "open" cases, gives the impression that possibly too little attention has been paid to the condition.

The *incidence* of the disease in the dog, as given by different authors, varies. Gaiger and Davies (1946) stated that the incidence was about 1 to 5 per cent. in different countries; Innes (1940), in a survey of the literature, gave figures from 1 to 13 per cent. (these figures were for dogs dying in clinics in France, Germany, Holland and Denmark); Martin (1946) in U.S.A. gave the incidence as 0·25 per cent. in a series of 3,000 dogs; Crocker (1919) for a similar number of cases gave the incidence as 1 per cent.; Feldman (1934) said that "the disease may be more common than thought, since the salient features are not recognised either during life or at *post-mortem* examination." He cited Douville, who is said to have given the incidence of tuberculosis in dogs in France as being 4 per cent.

The incidence here in Liverpool was 6·8 per cent. of a total of 165 dogs autopsied. There must obviously be great variation in the incidence in different areas and in cities compared with rural areas. It may well be that the high incidence encountered here bears a close relationship to the proximity of Cheshire with its high incidence of bovine tuberculosis. This suggestion receives support from the high incidence of tuberculosis in cats in Liverpool. Unfortunately, very few cats ever come to *post-mortem* examination, so that only rough estimates of the frequency of the disease can be given. Dobson (1930), in a series of 505 feline autopsies, gave the incidence as 2·1 per cent.; Innes (1940) said the incidence in Continental clinics varied from 0·2 to 7·0 per cent. In the cases here presented, 100 cats were examined and 13 (13 per cent.) were found to be infected with tuberculosis. This is an extremely high figure and the figure becomes of greater significance when, as will be seen later, most of these cases appeared to have arisen through the ingestion of tuberculous milk. It would be of interest to determine the incidence of bovine tuberculosis in young children in this same area.

The average age of the affected dogs was five years. Cats became affected at an earlier age, the average being about two years. In the dog no marked breed or sex susceptibility was noted but an impression was gained that the larger dogs were more commonly affected.

* Presented to the Lancashire Division, N.V.M.A., at Manchester, April 6th, 1949.

The *type* of tubercle bacillus was determined in seven of the eleven dog cases. Typing was carried out by cultural examinations and by animal inoculation. Four were of the bovine type and three of the human type. Stableforth (1929), in a total of 16 cases, found ten to be of the human and six to be of the bovine type. Moltzen-Nielsen and Plum (1943), in a survey of the literature, noted that 70 per cent. of cases were human in type, 27 per cent. bovine, 0·5 per cent. avian and 2 per cent. atypical. The infection in the cat is said to be invariably of bovine origin. In six cases only in this series of 13 was typing carried out and in each case the bovine tubercle bacillus was responsible.

Pathogenesis

In attempts to determine the pathogenesis of tuberculosis a number of fundamental questions must be answered. These may be summarised briefly as :—

1. What is the portal of entry of the bacilli ?
2. What is the reaction of the host to this invasion ?
3. How is the disease spread inside the host's body ?

Dealing with portal of entry, it is at once obvious that there are two main channels of entry, the respiratory tract and the alimentary tract, but each of these two routes has been the subject of much discussion and as yet the question is unanswered. The route of entry will decide the site of localisation of the bacilli and consequently the formation of the primary lesion. The site of this primary lesion then determines (in conjunction with other factors to be discussed) the rapidity or otherwise of the local spread by direct extension. Some tissues appear to be unsuitable to this development. The site of this primary lesion, once it is established, is of importance in (*a*) its proximity to vital organs and (*b*) its relation to secondary spread. Both these factors will have a direct effect upon the type and duration of the disease. It will be apparent, with these points in mind, how pre-eminently suitable, from the viewpoint of the tubercle bacillus, the aerogenous route of infection may be.

The bacilli on gaining access to the tissues must multiply and thereby produce disease, remain quiescent for some time, or be destroyed by the tissues and fluids of the host. Which of these states will obtain will depend upon the interplay of a number of factors. These include the number and the virulence of the invading tubercle bacilli. By virulence is meant, roughly, the ability of the organisms to multiply, the ability of the bacilli to produce toxins, and their ability to resist phagocytosis. Against these must be set the resistance of the host which in a broad sense implies the effectiveness of the tissues in their aggressive properties and the effectiveness of phagocytosis. The result of the reaction between all these most variable factors will determine whether or not the animal will develop tuberculosis.

Tuberculosis in any animal begins with what is termed a primary complex. If the tubercle bacilli multiply in the site in which they have been deposited a primary focus of disease is produced and in a very short time the regional lymph node which drains the area becomes infected. Thus is produced a primary complete complex. In some animals this is not always the case and an incomplete primary complex may be formed. This is assumed to arise by the silent passage of the bacilli through a mucous surface and then a localisation and multiplication in the regional lymph node. Incomplete primary complexes are common in the cat where the ileo-colic lymph node may be grossly diseased with no apparent change in the bowel. A possible explanation of this may be that the bacilli are phagocytosed and carried through the mucosa to the nearest lymph node ; there they may succeed in killing off the phagocytic cell and so disease is produced in the gland.

Having produced a primary lesion the disease will now spread through the body of the host by one or more of the following methods.

(1) By direct extension of the disease process into the surrounding tissue.

(2) By mechanical spread. This is probably of great importance in the pulmonary disease of the dog. Infected material is spread by aspiration and gravitation of infected fluid through the lung tissue. In a similar fashion the bladder and the ureters may be infected from a tuberculous kidney.

(3) By lymphatic spread, the tubercle bacilli have a predilection for the lymphatics and much of the spread in the body takes place in this way. The disease extends progressively from lymph node to lymph node.

(4) By the blood stream. It is through the medium of the blood stream that widespread dissemination of the disease takes place.

In the cases about to be presented the criteria of primary infection adopted were :—

(*a*) Where the lesion was the only one in the body ;

(*b*) Where a lesion was thought to be the oldest in the body, was localised, and was in a situation which was a possible portal of entry into the body.

Lesions were judged as acute or recent if, on microscopical examination, they showed no evidence of encapsulation, a rapid coagulative necrosis with local infiltration and a well-marked epithelioid cell reaction. The reaction of the host to invasion by the tubercle bacilli was a combination of cellular and humoral factors. The exudative type is especially well marked in the dog and is responsible for some of the well marked clinical symptoms.

Lesions

The gross appearance of the lesions varies somewhat. Generally, tuberculous lesions in the dog resemble neoplasms, especially the sarcomata, and it is probable that many cases are wrongly diagnosed during hasty *post-mortem* examinations. Often the tubercles are greyish white in colour and are circumscribed. The large liver lesions are yellowish with depressed centres and crenated haemorrhagic edges. Some of these lesions have soft almost purulent centres. In some cases the lesions appear as ragged bloody cavities, in others the hepatic lesions take the form of small multiple grey nodules scattered throughout the liver substance.

The lung lesions, in both species, take the form of greyish-red broncho-pneumonic areas ; some, especially in the cat, break down with the formation of cavities. These cavities may open into the pleural cavity or communicate with a bronchus. Early spreading lesions may vary from areas of acute congestion to hepatisation. These pulmonary and pleural lesions are invariably exudative in type and there may be pints of a straw-coloured liquid in the chest. This may give rise to pressure collapse of the lower portions of the lung. Renal lesions take the form of either single or multiple infarcts, the majority of which are found in the cortex. They are wedge shaped and grey in colour. They may be so small as to be visible only on microscopical examination. Intestinal lesions consist essentially of ulcerated Peyer's patches with small submucosal tubercles. Almost invariably they are secondary to extensive lung lesions and arise by swallowing heavily infected sputum.

Ulceration of the skin of the face, neck and fore-limbs is not uncommon in the cat. These skin lesions may be seen as flat red ulcers or as soft swellings with muco-purulent centres. They arise by the infection of wounds, cat bites, etc., by tubercle bacilli present in the nasal and pharyngeal exudate in open pulmonary cases of tuberculosis.

Finally, a condition affecting the bones must be mentioned since it may be seen in cases of tuberculosis although it is not tuberculous in origin. This condition has not been encountered in this series of cases of tuberculosis but has been seen in a case of multiple carcinomata in a dog. The animal had extensive metastases in the lungs. The condition has had several names applied to it, *e.g.*, Marie's disease, acropachia, and hyperplastic periostitis. It is characterised by an overgrowth of bone from the periosteum which becomes covered by bony protuberances. The limb bones are chiefly affected.

The microscopic appearances of the tuberculous lesions seen in this series of cases will not be dealt with except to point out that the disease is essentially a rapid one with extensive necrosis and caseation; rarely does calcification occur. The necrosis is undoubtedly responsible for the rapid spread of the disease through the lung and for the formation of *vomicae*. These arise as the soft necrotic centre of the tubercle is removed by phagocytosis or expectoration and the lesion then breaks down.

Distribution

The distribution of the lesions in the dog may be seen in Table I. In two of the cases (6 and 12) the lesions were confined to the thoracic cavity and in two (19 and 22) lesions were present only in the abdomen. The remaining seven cases had lesions in both cavities. The organs most frequently affected were lungs (eight), mesenteric lymph nodes (seven), pleura and liver (five), pericardium (four).

Table I.—Distribution of Lesions in the Dog

Case No.	Type	Lungs and adjacent lymph nodes	Pleura	Pericardium	Myocardium	Liver	Mesenteric lymph nodes	Kidneys	Uterus	Skin	Other lymph nodes
2	B	+	+				+				
3	B	+					+			+	+
4	B	+				+	+	+			
6	H	+	+	+							
8	H	+				+					
9	B	+	+	+		+	+				
11	—			+	+	+					
12	H		+								
19	—						+				
22	—						+				
23	—	+	+	+		+	+				

Individual Cases

Dealing now with the individual cases, one case, No. 3, had an unusual route of infection but it was one which was fairly definite but not beyond dispute. The subject was a six-months-old Great Dane bitch. The primary lesion was an extensive tuberculous granuloma which arose in the side of the neck following a subcutaneous inoculation. The dog had received an inoculation and this was followed by a suppurating swelling which did not resolve. Bovine tubercle bacilli were isolated from the lesion and circumstances suggest that the infection arose from the use of dirty instruments. The dog came to *post-mortem* examination and the distribution of the lesions was then noted. The disease had spread to the local lymph nodes (the cervical chain and the prescapular gland) and had then gained access to the lungs through the blood stream. These visceral lesions were very recent as compared with the skin lesion.

Cases Nos. 6 and 12 had lesions which were confined to the thoracic cavity and infection was almost certainly aerogenous. There was present in each case a complete primary complex with lesions in the lungs, largely sub-pleural, and enlarged caseous bronchial lymph nodes. Case No. 8 was thought to be of respiratory origin with secondary haematogenous spread to the liver. These liver lesions were multiple and diffuse. In this case there was a well-defined respiratory primary complex. Case No. 23, a Bull Mastiff of eight months, and No. 9, a Terrier aged four years, were also thought to be of respiratory origin. The first of these, No. 23, had an acute broncho-pneumonia and a pleuritis with copious serofibrinous exudation. However, there were lesions in the cervical chain and in the mesenteric lymph nodes and it is presumed that these arose by ingestion of infected sputum (there was a history of persistent cough). The other dog, No. 9, was an open case with several vomicae present and the lesions in the liver may have been haematogenous or alimentary in origin, probably the latter since the mesenteric glands were involved. The swallowing of infected sputum would explain these lesions.

In two cases, Nos. 19 and 22, the lesions were confined to the abdominal cavity and infection was therefore assumed to be alimentary. In both cases there was an incomplete primary complex; the ileocolic lymph nodes were grossly diseased.

The remaining three cases were extremely difficult to assess, especially case No. 11. This was a mongrel dog of about four years. There were present multiple extensive heart lesions which involved the complete heart wall, mainly the auricles, and there was extensive pericarditis. Careful search, both macroscopic and microscopic, of the lungs failed to reveal any lesions. No lesions could be found in either the bronchial or the mesenteric lymph nodes. The liver of this animal contained about 15 large yellow tubercles which were thought to be older than the heart lesions. The main clinical feature of this case was the very marked ascites, several pints of an opalescent fluid being present. Cases Nos. 2 and 4 had tuberculous lesions in both body cavities. No. 4, which showed involvement of the liver and kidneys, had definitely had secondary haematogenous spread but no opinion is expressed as to the route of infection.

It was concluded, therefore, that in these eleven cases of canine tuberculosis five were of respiratory origin, two were alimentary and in three the route of infection was not determined. In one case infection was by subcutaneous inoculation. These findings resemble those of other workers. Innes (1940) in eight cases had five which were said to be of respiratory origin. Nieberle (cited by Innes, 1940) had 28 out of 33 which showed a complete or incomplete primary pulmonary complex. Stableforth (1929) in a series of 16 cases described ten of them as of respiratory origin. Of the remainder one was definitely alimentary in origin, one probably pharyngeal and four uncertain.

Distribution of lesions in the *cat* may be seen in Table II. The organs most frequently affected were as follows: Lymph

Table II.—Distribution of Lesions in the Cat

Case No.	Type	Lungs and adjacent lymph nodes	Pleura	Pericardium	Myocardium	Liver	Mesenteric lymph nodes	Kidneys	Uterus	Skin	Other lymph nodes	Other organs
1	B						+		+			Mesentery
5	—						+					
7	B	+					+					
10	B						+					
13	B	+				+	+					Spleen
14	B	+					+	+	+			Mesentery
15	—	+					+					
16	B	+	+	+								
17	—	+	+				+			+		Retropharyngal lymph nodes
18	—	+					+					Mesentery
20	—						+					
21	—	+					+	+	+			
24	—						+					

nodes (twelve), lungs and adjacent lymph nodes (eight), uterus (three), kidneys (two), pleura (two).

The Route of Infection

Dealing now with the route of infection, it will be seen at once that there is a difference in the two species. In one cat only, could a respiratory route of infection be traced. In this case, No. 16, the lesions were confined to the thorax. The initial lesion appeared to be in the left apical lobe but both lungs showed areas of broncho-pneumonia which varied in severity. The lymph glands in the mediastinum were grossly enlarged and formed one large fused mass. There was extensive pleural effusion and this had brought about collapse of the ventral borders of the lungs. The pleura and the pericardium showed red velvety granulations and were thickened.

Case No. 17, in which the lungs and pleura were extensively involved, was presumed to be due to infection in the naso-pharyngeal region. The lymph nodes of this area were grossly infected and consisted of little else but a semi-solid mass of necrotic tissue. The lung lesions probably arose by aspiration of infective material from this area. A large granuloma was found in the skin and subcutis halfway down the neck. There was said to be an old cat bite at this site. Tuberculous animals are prone to infect wounds or bites on their skin by licking the affected parts, and this is a potential source of danger to humans in contact with them.

The remaining eleven cases were clearly of alimentary origin. In each case there was an incomplete primary complex. The mesenteric lymph glands usually were extensively involved but in no case was involvement of the bowel mucosa noted, despite the fact that in some of the cats the grossly enlarged lymph nodes and loops of bowel formed a single mass. Invariably the ileocolic gland was involved and it appears as if this is the place in which the first visible lesions develop. The pulmonary lesions arose by spread from these glands *via* either the thoracic duct or the venous circulation or both. The spread then became haematogenous. Direct spread from the colic gland to the peritoneum occurred in case No. 14. This animal showed a most extensive peritonitis with ascites. It is probable also that the uterus may have been infected by direct spread since there was extensive tuberculosis of the uterus throughout its length.

Previous workers recorded similar findings; thus Dobson (1930) found that ten of eleven cases of feline tuberculosis were alimentary in origin. Griffith (1926) had ten out of eleven, Stableforth (1929) four out of five and Innes (1940) 13 out of 13. This alimentary route of infection is not agreed upon by some Continental workers; Nieberle stated that in 18 cases of tuberculosis in the cat 13 of them had a primary pulmonary infection.

A comparison of the lesion distribution in the two species shows the results to be what one would expect, having in mind the somewhat different portals of entry of the tubercle bacilli. In the dog infection is largely *via* the respiratory tract whereas in the cat it is almost invariably through the intestinal tract. Experimentally it is not easy to infect these animals and a variety of routes of inoculation has been tried. Apart from the local spread, however, *i.e.*, from the primary lesion which will be directly governed by the route of inoculation, it appears as if the tubercle bacilli have predilection sites in different species regardless of the mode of infection. This organ susceptibility has been noted by many workers. Even from the few cases presented here some such selectivity may be noted. In an effort to determine some of the factors which may influence this predilection Corper and Lurie (1926) carried out a series of experiments on dogs. They used the intravenous route as the one most easily controlled and one which assured access to all organs. They noted that the lungs of the dog provided an excellent soil and that relatively few bacilli were needed to colonise it, whereas the spleen was rarely involved and even after massive numbers of tubercle bacilli had been given small lesions only developed—this despite the fact that the spleen contained large numbers of bacilli. It would appear as if the spleen of the dog may contain an inhibitory substance. Similarly in the rabbit the liver may show no lesions and yet contain large numbers of organisms. These workers also showed that the localisation of the tubercle bacilli followed closely the localisation of injected carbon particles, *i.e.*, that the same phagocytic cells were involved. The distribution of the bacilli in the body corresponded roughly to that of the reticulo endothelial system. The exact reasons why certain organs appear to be inimical to the tubercle formation are still unknown. Probably the answer lies in cell metabolism since it has been shown that certain chemical fractions of the tubercle bacillus induce specific effects on cells. The waxes are said to be responsible for the conversion of the tissue histiocyte to the typical epithelioid cell of the tubercle. The tuberculo-proteins amongst other things stimulate plasma cell migration and the polysaccharides exert a similar effect upon the leucocytes. Whether or not disease will be set up will depend finally upon the result of the interaction of two metabolic processes, that of the invading organism and that of the host cell.

To return to the cases under discussion, it will be seen that the liver of the cat does not appear to be a suitable medium for the tubercle bacillus, whereas that of the dog is most suitable. The same point was noted during the survey of

other cases in the literature. The uterus does appear to be a suitable organ since in five female cats there were lesions of tuberculosis in three of them. The possibility of congenital infection in the cat becomes at once apparent. In one case the cat had had kittens some eight weeks previously; unfortunately these were not available for examination. The lesions in the uterus were well formed and there had been a history of a continuous vaginal discharge since parturition. This exudate contained innumerable acid-fast bacilli. In this connection it is interesting to note that Griffith (1926) described a case of tuberculous choroiditis and detached retina in a kitten, the survivor of a litter, which had been destroyed for blindness. A litter mate had also been destroyed for blindness. Griffith does not state what the lesions, if any, were in either the others or in the dam. It is suggested that the possibility of the spread of infection from the uterus be borne in mind. The kidneys of the cat are also frequently involved and many of these cases are "open" ones. Examination of the urinary sediment may reveal tubercle bacilli.

Serous membranes in general appear to be more often involved in the dog than in the cat, as also do the lungs and heart. It is realised that with so few cases only impressions may be gained. It is proposed therefore to end this paper by citing the work of Bonaduce (1942) on the predilection sites in the dog and cat. This worker surveyed the literature on canine and feline tuberculosis from 1882 to 1940, and his findings may be summarised briefly as follows. Pleural tuberculosis was recorded in 40·74 per cent. of tuberculous dogs, but was present in only 6·51 per cent. of infected cats. Liver, kidney, heart, intestinal and nervous lesions were said to be more common in the dog. (The inclusion of intestinal lesions in this group is surprising.) In the cat, spleen and skin lesions occurred more frequently than in the dog. Bone, joint and genital lesions were said to be three to four times as common in the dog as in the cat. From his survey Bonaduce concluded that some morbid processes seem to be exclusive to one or other species, for example, hypertrophic osteoperiostitis in the dog and various oculo-conjunctival lesions in the cat.

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Opening Contribution to Discussion

Mr. A. C. Shuttleworth, F.R.C.V.S.: We must congratulate Mr. Jennings and thank him for giving us this clear and detailed paper upon the rather neglected subject of tuberculosis of the small animals. He has used the material at his disposal usefully and I would ask the practitioner to help in this matter of pathological material for investigation. All such help will be eagerly seized to combat this disgusting disease of the human and animal world. Tuberculosis of the dog and cat is but a small subject in the grim, grey picture of the disease among humans and the farm animals. Nevertheless, it is the disease in a peculiarly tragic form—illness and ultimate certain death being passed unwittingly between those good friends, man, dog and cat.

The incidence, as Mr. Jennings has mentioned, varies from different sources quite widely from a fractional percentage up to 13 per cent. The incidence in the clinic at the London Veterinary College has been calculated by Smythe at between 0·3 per cent. and 0·4 per cent. of the total number of dogs submitted for treatment. Mr. Jennings finds the percentage as high as 6·8 per cent. for dogs and 13 per cent. for cats. The Department of Veterinary Pathology at Liverpool receives the majority of our interesting and obscure cases, and similarly, such cases from other sources, so I admit the high percentage mentioned as referring to these selected cases. I do not think Mr. Jennings intends us to understand that six dogs in every 100 or 13 cats in every hundred are sufferers from tuberculosis. I consider that 0·6 per cent. of cats and 0·15 per cent. of dogs admitted to our clinic are tuberculous.

So low is the incidence, that tuberculosis suspicion in the clinic comes low in the scale for consideration—far behind chronic nephritis and malignancy in the older dogs, or distemper and leptospirosis in the younger animals. I have found that suspicion of tuberculosis becomes a periodic "bee in the bonnet." Following the discovery of such a case, every vague and doubtful case comes under suspicion. This conjectural frame of mind slowly subsides until another typical case turns up, when the bee commences to buzz again.

The average age of the affected dog appears to be five years, and so far as I have been able to find, with the exception of Gray (1931) no one mentions the disease in the very young puppy. There is apparently a well-defined resistance to tuberculosis in the dog which may become less or be overborne in later life.

There is an insistence from a number of observers that it is not the small pet dog that suffers, but the mid-sized or large animal, and such is our experience. One would consider that the lap dog with his closer human contact would be the chief sufferer from the human, air-borne infection. In the same way, with his fondness for the human lap, the cat would appear to be menaced by the human infection, but he rarely is. I suppose the frequent licking of the owner's face is a common source of transfer of the disease; the cat rarely shows his affection in this way.

There are well-defined manifestations of the disease in the dog during the later stages of the disease—increasing tiredness and weakness, emaciation, sunken eyes, due to the loss of the post-orbital fat, and a resultant sad expression. Together with these general symptoms, the local evidence may show thoracic affection with rapid and laborious respiration suggesting hydrothorax and lung collapse. Coughing may be present and auscultation of the lungs may show râles and areas of dullness. Abdominal symptoms, in the characteristic form, show a degree of ascites and the abdomen may become very pendulous due to the great amount of fluid present. Palpation of the abdominal wall may reveal a massing of the structures indicative of some grave change, possibly tuberculosis, possibly malignancy, but certainly hopeless. In some cases the edge of the enlarged and tough lobes of the liver may be felt projecting well beyond the costal margin.

In the case of the cat we find the usual form of the disease to be abdominal. It is seen most frequently in the young adult and the early symptoms are indefinite with slight malaise, inappetence and perhaps occasional vomiting. The animal has not lost flesh and the disease is certainly not acute at this stage. Nevertheless, the abdominal lesion is frequently obvious in the form of a distinct or great enlargement of the ileo-colic lymph gland. Its hardness and nodular surface will not be confused with an impacted colon or the dependent left kidney. If the cat is seen later in the course of the disease the emaciation is evident; the abdominal mass is increased in size by the matting together of lymph gland. Omentum and intestinal loops. Ascites may be present. Such an array of symptoms may be considered to be due to a lympho-sarcoma. Thoracic symptoms are not common in the cat, unless in conjunction with the abdominal form, when they are manifested as hydrothorax.

Mr. Jennings has mentioned an irregular case in a Great Dane puppy. This young bitch had suffered from distemper with pneumonia ten weeks previously and had received inoculations at this time. A large swelling occurred at the site of injection on the side of the neck which, following poulticing, had opened as an abscess but continued to discharge pus and increase rapidly in size. When brought to Liverpool the swelling was extreme, reaching from the jaw to the shoulder and pitted by numerous sinuses.

A sarcoma was considered and possibly some gross infection. Swabs taken for examination revealed acid-fast organisms and the dog was destroyed. The rapidity of the spread through the protective lymph nodes and into the general circulation is of interest. This infection may have been due to a soiled syringe or needle, or later infection of the inoculation wound. Such a case was, of course, extremely dangerous to the owners. Other open cases are met in the form of the suppurating tracks leading from the retro-pharyngeal glands. We have attempted removal of such infected lymph nodes but the chronic infection produces such a conglomeration of fibrous tissue entangling the jugular, carotid and other structures, that the operation is very difficult and re-infection of the wound certain. Abscess formation due to other organisms, cystic new growths and malignant neoplasms are not rare in the neck region, but persistence of sinus formation should be checked for the presence of acid-fast organisms. As in other open cases destruction is to be advised. In the case of the cat, ulceration of the upper lip, at the philtrum, with erosion of one or both sides of the cleft is sometimes seen and is regarded as a tuberculous dermatitis. This is not always so but should be ascertained microscopically. In the cat, again, Mr. Jennings has illustrated another method of infection. The animal was said to have suffered a bite or other injury in the neck which developed into an acute tuberculous granuloma. It is suggested that the bite wound was infected by the cat while licking, as he was found on *post-mortem* examination to have a tuberculous lesion in the naso-pharyngeal region. Almost certainly this cat would implant his opponent with the disease in some part of his anatomy, and it behoves us to watch with care these persistent sinuses of the limbs and other regions of the cat following fights. For some time past the Department of Pathology has been examining smears from all such cases presented at Liverpool, but so far no acid-fast organisms have been seen. The open thoracic lesion in the dog and cat, accompanied by a chronic cough, is a distinct threat to the owners and other animals, but it is remarkable that with the increased attention to the bacterial flora of the pharynx which the fear of *Beta haemolytic streptococcus* has aroused, no direct diagnosis of tuberculosis has been made, in our experience, from this source.

From the evidence placed before us this afternoon it is apparent that the dog and cat are not easy victims to the disease, and we can anticipate a fall in the incidence as the campaign against bovine and human tuberculosis succeeds. In the meantime, the aim is to determine at the earliest moment the open cases by means of smears and bacterial examination and, when confirmed, destruction. For closed cases an early diagnosis from the clinical evidence, together with subcutaneous or intradermal testing, is necessary, and destruction in these cases also.

THE FUTURE OF AFRICAN GAME

The growing threat to African game animals, says *The Field* in a recent issue, has been put into its right perspective by Dr. D. G. Davey, one of the discoverers of the new drug Antrycide, the latest weapon in the war against the tsetse fly. Speaking to a joint meeting of the Royal Empire and Royal African Societies, he said: "Freedom for African game and the development of Africa are incompatible ideas. As development goes on it will push the wild life before it. If we want that development we must accept the fact that the wild animals will become fewer, but we should also accept as another fact that it is our duty to see that they are not annihilated, that a home for these majestic and graceful creatures should be found in reserves where they can live their lives in freedom."

"This supports the views expressed in *The Field* more than once," observes that periodical, adding: "The question remains as to how and where game reserves can be established. The need for an international commission to make recommendations is suggested. But action of some sort is clearly an urgent matter."

Because of the special circumstances under which the Congress is being held in London, and in view of the paramount importance of the theme of the Congress, *i.e.*, the veterinary profession's contribution to the world's food supply, we must make the XIVth International Veterinary Congress the most successful ever held.

THE VETERINARY RECORD
July 2, 1949

XIVth INTERNATIONAL VETERINARY CONGRESS

NOW that the date of the International Congress is drawing near it may interest some of our readers to know how the idea of the International Veterinary Congress originated.

Eighty-five years ago, at the suggestion of a distinguished British veterinarian, John Gamgee, the First International Veterinary Congress was held. At that time the prevalence of cattle plague (rinderpest) over a large part of Europe was causing grave concern. Professor Gamgee recognised that the control of animal plagues could not depend entirely upon the individual efforts of each country, but must be made the common interest of all. The wisdom of the suggestion was recognised immediately by all the European countries. In the course of time the field of discussion has been so widened that it now embraces every sphere of veterinary interest. The Congresses which have been held have materially assisted the advancement of veterinary science.

The International Veterinary Congresses normally are held every four years, and have been held in many countries, the last having taken place in Switzerland in 1938. In 1914 the Congress was held in London, but the 1914-18 war was declared immediately after the Congress opened and it had to be abandoned. London was again the venue in 1930, and under normal circumstances it would not have been held again in Britain for many years to come.

Our readers will have noted in the programme which was published in THE VETERINARY RECORD of May 7th, that papers are being submitted from many countries, and the contributions are, therefore, truly international.

Enrolments for membership are coming in well from abroad. The Netherlands and the Scandinavian countries will be particularly well represented, and also a large contingent from the U.S.A. is confidently expected.

One or two major changes have had to be made in the programme as previously published; in particular, Dr. P. J. Du Toit of South Africa and Dr. F. R. Beaudette of the U.S.A., who were to have given two of the principal lectures on the world's meat supply and the world's poultry and egg supply, respectively, have since found that it will be impossible for them to attend. The Programme Committee have been most fortunate in getting Dr. C. S. M. Hopkirk of New Zealand and Dr. J. R. Beach of the U.S.A. to take their places at rather short notice.

We are glad to be able to publish a time-table of events in this issue. Special attention may, perhaps, be drawn to Sunday, August 7th, the day before the Congress opens, when tea and conversazione will be held in Ashburnham House and Westminster School from 4 p.m. to 6 p.m., and by kind permission of the Dean of Westminster, members of the Congress may participate in a special form of evensong at Westminster Abbey at 6.30 p.m. This service will be attended by the President and Council of the Royal College of Veterinary Surgeons and the President and Council of the N.V.M.A. and other interested bodies will be officially represented. It is certain that many British veterinary surgeons and their ladies will attend. It will facilitate the work of the Congress if as many members as possible will register on Sunday afternoon in order to prevent unnecessary congestion and delay at the opening session on Monday morning.

CLINICAL COMMUNICATIONS

OSTEOMALACIA IN PREGNANT JERSEY HEIFERS

H. F. HEBELER, M.R.C.V.S.

LANGPORT

Subjects.—Two pedigree Jersey heifers. One, named "Blackmore," was born in October, 1946, and served in late March, 1948; the other, named "Drake," was born in February, 1947, and served in late May, 1948.

History.—On December 5th, 1948, the heifer "Blackmore" was seen to be slightly lame and closer inspection revealed an abnormal posture of the forelegs. Four days later the second heifer was found to be in the same condition, the onset of which must have been very sudden as a careful inspection of the group had been carried out daily following the first case.

Clinical Signs.—Outstanding was the gross "bowing" of the forelegs, the condition being centred in the epiphyses of both radii. The distal epiphyses were more obviously affected, being enlarged, somewhat tender on firm handling and showing an apparent "angle" at the point of abduction. The gait was stiff and awkward, especially in the heifer "Blackmore," which appeared to feel pain if moved any distance. The deformity caused nearly all the weight to be taken on the lateral edge of the sole of the lateral claw. Blood samples taken from all the heifers in the group 16 days after treatment had been started revealed no abnormality except a tendency to low haemoglobin values. (*Report appended.*)

HEIFER A.—"BLACKMORE."

HEIFER A.—"BLACKMORE."

HEIFER B.—"DRAKE."

Normal From Same Batch.

Diagnosis.—Osteomalacia, probably due to calcium deficiency of unknown etiology.

Treatment.—The affected animals and the other six heifers in the group (all about the same age and at the same stage of pregnancy) immediately were fed a small ration of dredge corn with the addition of 1 fl. oz. of cod liver oil and 1 oz. of best quality steamed bone flour (Dunlop's) daily. The remaining animals over six months of age were fed a good quality mineral supplement with a bone flour base. The two affected animals were housed in an effort to encourage recumbency and so take the weight off the radii ; this proved successful and the deformity did not become more marked. Both animals adjusted their gait to the condition and both calved normally at full term, neither of the calves has shown any abnormality to date nor has any other heifer in the group shown any further symptoms.

Discussion

A summary of the herd and farming methods is as follows: The farm consists of 43 acres at an altitude of 100 feet ; the soil is medium lias clay on limestone and, in many places, only five or six inches deep. About 40 head of pedigree attested stock are kept on intensive lines ; hay and concentrate cakes are purchased. Two to four calves are reared on a nurse cow from four days after birth until old enough to feed really well on meal, milk powder and hay with cod liver oil added daily.

In the spring the calves go out, by day only at first, on to one or other of two pieces of permanent pasture. In the autumn and winter they are moved to another piece of permanent pasture and given kale and oat straw, replaced by hay later. In the summer the heifers, now pregnant, follow the dairy cows in grazing the leys on intensive lines controlled by electric fencing. During September, 1948, the group in question grazed a piece of lucerne-cocksfoot mixture for three weeks before returning to the leys for as long as they remained sufficiently dry, kale and oat straw being fed later ; normally no concentrates are fed to the heifers until just before they are due to calve.

Soil samples are taken regularly from each field and the land is dressed as indicated. Since 1944 there has been a steady rise in the calcium and phosphorus content of every field in question.

In attempting to elucidate the etiology of this condition two facts are outstandingly apparent:

1. The early age at which these heifers are required to conceive.
2. The intensive grazing methods in which the pregnant heifers follow the dairy cows.

Beyond these facts, and allowing for the coincidence of individual idiosyncrasy, the etiology is obscure. Two conjectures can be made:

1. The calcium and phosphorus content of the herbage in intensively grazed leys is not a reflection of the content of the soil.
2. The calcium and phosphorus content of such herbage, if present, is in a form not so readily assimilated by the animal as in other foodstuffs.

Acknowledgments.—Thanks are due to Mr. D. W. Menzies, V.I.O. Bristol, for his prompt help and suggestions in this case ; and to Dr. H. H. Green for his report on the blood samples.

Biochemical Department Report—V.L.401

Chemical Data on Eight Sets of Blood Samples from Cows belonging to Colonel Bray sent by Mr. H. F. Hebeler, M.R.C.V.S.

Name	Mg. % serum			Mg. % blood		G. % blood
	Calcium	Magnesium	Acetone	Sugar	Inorganic P.	Haemoglobin
Blackmore	10·3	2·5	Normal	62	4·7	8·3
Diana ...	10·5	2·2	„	60	4·3	9·2
Drake ...	10·4	2·5	„	58	5·3	8·6
Erebus ...	10·1	2·3	„	64	4·8	8·5
Gloucester	10·1	2·4	„	55	5·0	8·8
Joanna ...	11·0	2·5	„	58	4·5	9·8
Severn ...	10·5	2·4	„	58	3·9	8·6
Templar II	10·4	2·4	„	58	4·4	8·6

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STRANGULATED INGUINAL HERNIA IN A MONKEY

J. B. EDWARDS, M.R.C.V.S.

Wellingborough

Subject.—A "Sooty Mangaby" male monkey, about 8 years of age.

History.—Had shown great discomfort for 24 hours, passing small quantities of urine frequently. Owner noticed a swelling in the groin.

Examination.—The monkey was anaesthetised, using a dog-size ether-oxygen apparatus. A tense swelling was observed in the left groin ; the penis was swollen. The swelling was doughy to the touch, did not fluctuate and could not be reduced.

Diagnosis.—Strangulated inguinal hernia.

Operation.—Operation was performed immediately.

The site having been prepared, the skin was incised and the hernial sac was separated by blunt dissection. It was suspected to contain a loop of bowel and was some 2½ inches in length. The sac was opened along its length and the contents revealed were, firstly, a large piece of very congested mesentery which was drawn out until healthy tissue was seen ; it was then ligatured and removed. It now became possible to return the healthy portion to the abdomen and the remaining contents of the sac could be seen. These were the bladder and the left testicle. It was necessary to enlarge the inguinal ring in order to return the bladder. The testicle was then removed, the sac was ligatured at its base, the ring and incision closed with gut sutures and the skin incision closed with nylon halstead sutures. An injection of 5 mega units penicillin was given.

After-Treatment.—The animal was confined in a small cage for three weeks, the sutures being removed 10 days after the operation. Healing was by first intention and recovery was uneventful.

It would appear that in these animals the testicles are carried in a position anterior to the penis, thus simulating, so I am informed, the partially descended testicle which occurs in humans. The bladder is occasionally met as the herniated organ in humans, but I can find no record of this having occurred in animals.

ABSTRACTS

[Foot Rot in Cattle : A Small Controlled Experiment. Roberts, S. J., Kiesel, G. K., & Lewis, N. F. (1948.) *Cornell Vet.* **38.** 2.]

The literature on the sulphonamide therapy of foot rot in cattle is briefly reviewed.

The experiment was undertaken because the sulphonamide treatment of foot rot does not follow the accepted therapeutic rule for sulphonamide therapy, in that effective blood levels are maintained for only six to twelve hours following a single intravenous injection.

136 cases of foot rot in 56 herds were treated thus :— 13 cases received 60 to 90 grammes sod. sulphapyridine intravenously. 17 cases received 50 to 80 grammes sulphapyridine orally. 37 cases received 60 to 80 grammes sod. sulphamerazine intravenously. 38 cases received, 60 to 80 grammes sulphamethazine intravenously. 31 cases received 500 cc. of 40 per cent. glucose solution intravenously.

Glucose was used because it had no therapeutic value in the disease, and because the solution had the same colour as the sulphonamide solutions. Thus roughly one-quarter of the cases were left as controls.

The owner was instructed in all cases to keep the cow confined in the byre for seven to ten days before returning her to pasture, and to inform the authors if there was no inprovement in three to five days. The necessity for retreatment was taken as a measure of the efficacy of the treatment.

The 105 cases receiving sulphonamide therapy showed a recovery rate from one treatment of 93.9 per cent., while the 31 controls receiving glucose showed a recovery rate of 80.6 per cent.

Details of cases failing to respond to one treatment are given.

In discussing the results the authors state that the cases responding to glucose did so just as rapidly as those receiving sulphonamide therapy. They consider that this may, and probably does, mean that most lesions of foot rot in cattle are self-limiting and will recover in five to ten days with rest alone. They state that in the years 1945 to 1946, 250 cases of foot rot were treated in their practice by the application of 20 per cent. ichthyol paste under a bandage with results as good as, or better than, those obtained later with the sulphonamides. It is emphasised that the use of drastic surgery is contra-indicated.

The authors consider that although the results with the sulphonamides showed a slight improvement upon the untreated controls, the difference was not sufficient to indicate that sulphonamide therapy is greatly superior to former methods of treatment.

A. M.

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[The Sedimentation Test and its Interpretation. Knowles, R. P. (1949.) *Vet. Med.* **44.** 50.]

The sedimentation test is based upon the observations of Fahraeus that if an anti-coagulant (3 per cent. solution sodium citrate) be added to whole blood the rate of sedimentation of erythrocytes will vary as between health and disease and in such physiological states as oestrus and pregnancy. The rate of sedimentation is measured by the depth in millimetres of clear plasma which is formed at the top of a vertical column of citrated blood estimated at fixed periods varying from 15 to 60 minutes according to the method employed. Blood for examination is diluted 4 parts to 1 of anti-coagulant and is then drawn into a glass tube with a bore of 2.50 mm. to a height of 60 mm. (Walton). The upper end of the tube is left open while the lower end is closed by a rubber cap held in position by a spring.

An alternative method is to place one drop of citrated blood on a tilted microscope slide.

The rate of suspension is an index of the suspension stability of the blood ; it is governed by the character of the plasma, not by that of the erythrocytes. It is thought that an increase in the fibrinogen and globulin fractions of plasma protein is responsible for the clumping of cells and their consequently more rapid rate of sedimentation (auto-agglutination).

The sedimentation test is non-specific, but to the clinician it is of value as an index to the progress, activity or resolution of an infection. It may be a guide to prognosis in that a progressive decrease in the sedimentation rate indicates a return of the organism to normal.

Since by far the greatest amount of information has been collected from human sources the following table of sedimentation rates expressed in millimetres per hour may be of interest :

Newly-born	0.5
Adult male	1 to 3
Adult female	4 to 7
Pregnancy	35
Pulmonary tuberculosis	65
Septicaemia	100

Regarded as a routine clinical aid to prognosis, Cutler postulates the following rates in millimetres per hour :

Normal	0 to 8
Moderate infection	8 to 18
Moderately severe increase	18 to 30
Very severe increase	30 upwards,

adding that a sedimentation rate above 44 mm. in the first hour almost invariably presages a fatal termination.

REVIEW

[Textbook of Meat Inspection. By H. THORNTON, B.V.SC., M.R.C.V.S., D.V.H. Pp. xi+656, 4-colour plates and 257 other illustrations. Price 50/-. Publishers Baillière, Tindall & Cox, 7 and 8, Henrietta Street, London, W.C.2.]

This book is a welcome addition to veterinary literature and our colleague is to be congratulated on producing a textbook which will close a long existing gap. Meat inspection has never been treated as seriously in this country as in America and on the Continent of Europe and for many years past reference has had to be made to foreign sources, especially to Ostertag whose classical work has been the " bible " of British veterinary meat inspectors.

Thornton's work is undoubtedly the best textbook on the subject that has emanated from this country and has been written not merely for the veterinary reader but for a much wider circle.

It is a very readable book yet it is concise and comprehensive, for it deals with the inspection of all the common food animals (including horses, poultry and fish), the inspection of eggs, refrigeration, preservation and canning and the utilisation of by-products.

The chapter on tuberculosis is perhaps the most useful from the student's view-point. The English and Scottish codes of inspection—which often are difficult for non-veterinarians to translate into logical and satisfactory practice—are examined and discussed in a manner which will meet with general approval, although some of the author's conclusions may not be accepted without reservation by some British veterinary meat inspectors—as, for instance, his view that the recommendation of Memo. 62/Foods that the entire carcase and organs shall be seized in cases of tuberculosis with emaciation, " would appear to apply to emaciation as a result of tuberculosis, and not to carcases which are emaciated and affected coincidentally with tuberculosis."

The chapter on bacterial diseases of food animals is well worth reading. The stress placed upon the desirability of employing routine bacteriological methods to supplement routine inspection of emergency slaughtered animals will meet with enthusiastic approval.

There are remarkably few typographical errors and inaccuracies, but it is a pity that many of the illustrations do not conform to the same high standard as the text. This is due, in some cases, to poor photography, in others to the reproductions being too small and in others probably to the fact that they would reproduce satisfactorily only on fine art paper.

This is a good book—a very good book—and both the author and publishers are to be congratulated on producing what will undoubtedly be acclaimed the most authoritative textbook on meat inspection so far issued in this country.

BILLS PASSED BY THE COMMONS

In the House of Commons on Friday of last week, The Slaughter of Animals (Scotland) Bill and the Cockfighting Bill (changed from Baiting of Animals Bill) were read the third time and passed.

Those wishing to read this subject more fully are referred to

WALTON, A. C. R. (1935.) *Quart. J. Med.* **2,** N.S., 79.
FAHRAEUS, R. (1921.) The suspension stability of the blood. *Acta Med. Skand.* **55,** 3; *Physiol. Rev.* (1929.) **9,** 241, and acknowledgment is made to Best and Taylor, *Physiological Basis of Medical Practice,* Third Edition, pp. 84-86.

QUESTIONS AND ANSWERS

The submission of questions for inclusion in this column will be welcomed; they can relate to any aspect of veterinary work. For purposes of record, each question is numbered, and those submitting questions are required to furnish (not for publication) name and address.

Answers to readers' queries represent exclusively the personal opinions of the writers, and criticism of the replies will be appreciated.

All communications should be addressed to the Editor.

Progeny Testing of Bulls and Boars

Q. CCCIX.—*To what extent is progeny testing of bulls and boars practised in this country? How many bulls at A.I. Centres are progeny tested?*

A.—At the present time, there are no official progeny testing stations for bulls in this country such as those in Denmark, where the daughters of the sire on test are kept under identical conditions. However, with the development in recent years of milk recording, the Bureau of Records of the M.M.B. is accumulating data which is available to breeders, breed societies and artificial insemination centres, and this will prove of great value in the progeny testing of bulls.

It is impossible to say how many progeny tested bulls are in use at artificial insemination centres, but it is unlikely to be more than 20 per cent. of the total number of bulls being used. Under conditions of natural mating, bulls are usually at least six years old before they become progeny tested. These older bulls are less likely to pass the necessary tests for artificial insemination than are younger bulls, so that of necessity artificial insemination centres have been forced to use a high proportion of young untested bulls.

However, as artificial insemination affords an opportunity for progeny testing at an earlier age than in natural mating, it will undoubtedly be found that as time goes on the proportion of progeny tested bulls in use on artificial insemination centres will increase rapidly.

As far as can be ascertained, there is no progeny testing of boars being done in this country at the present time.

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Aetiology and Treatment of Posterior Paralysis in Sows

Q. CCCX.—*Can you tell me the aetiology and best line of treatment of posterior paralysis in sows, occurring three to eight weeks after parturition? No other symptoms occur, the sow remaining apparently well in herself until death supervenes, usually in three or four days. The condition does not respond to treatment with calcium, glucose or strychnine.*

A.—The respondent presumes that by posterior paralysis is meant inability to raise the body with the hind legs, *i.e.*, sensory and motor function are not absent, but rather there is insufficient muscular power, similar in character to the condition of " loin drop " in cattle. The failure of calcium, glucose and strychnine therapy supports such an interpretation

There is little information available concerning such a syndrome in sows, but posterior paraplegias of varying severity occur in pigs in a variety of nutritional disorders, usually in deficient states associated with vitamins A and the B complex. It may also occur more rarely in certain uncommon infections, such as listerellosis, and as the result of traumatic injury either to the spinal column or the pelvic nerves during a difficult parturition.

It is not possible to rule out these possibilities with the data given. A full neurological examination, paying particular attention to possible defects in other parts of the nervous system, together with biochemical examination of the blood for Ca & P, would be most valuable. In the absence of a positive diagnosis, the best line of treatment would be good nursing, the feeding of brewers' yeast and

green stuffs or dried grass or lucerne of first quality, and the possible use of stimulants such as caffeine subcutaneously.

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Squamous Eczema in a Collie

Q. CCCXI.—*I should appreciate guidance as to treatment of a Collie, which seems to be losing its hair and has a squamous eczema. The condition definitely is not parasitic. The dog, a male, has had injections of antogenous vaccine, arsenic, sulphur baths, external resorcin lotions and thyroid tablets internally. He is four years old, fatter than he ought to be, and a cryptorchid. Also the descended testicle is smaller than normal.*

A.—Loss of hair and squamous eczema appear to be a not uncommon accompaniment of testicular dysfunction or subfunction.

Whether the thyroid is involved as well seems open to some doubt, although the writer has known such cases in young dogs give a response to a thyroid-whole pituitary mixture. (Suggested daily dose for Collie thyroid gr. i, plus pituitary whole gland, gr. i.)

This dose might be increased with safety, but a careful check should be kept on heart action if large doses are employed.

A more hopeful line of attack would appear to be:

(1) The administration of testosterone with or without thyroid/pituitary in addition.

(2) Surgical removal of the retained testicle, combined with a Steinach operation—ligation of vas deferens on the normal side with the object of stimulating testicular tissue to function.

Attempts to cause the retained testicle to descend by means of L.H. or other hormone stimulation are, in the writer's experience, unlikely to succeed.

Skin dressing may be confined to the simple relief of irritation, *e.g.*, calamine lotion plus liq. picis. carb.

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Examination of Greyhounds Before Racing

Q. CCCXII.—*What would you recommend as the most reliable method of examining each greyhound before its race? I am allowed approximately one minute per dog, including running-up. The average greyhound has a bad mouth and breath, and frequently congested eyes.*

A.—The officiating veterinary surgeon cannot pretend to examine every greyhound, before kennelling, to the same extent that, and with the same degree of care with which, he would examine a horse for soundness. Yet he can see much, and form a sound opinion in a short time if he is accustomed to greyhounds.

The guiding principles should be that he is reasonably satisfied:

(*a*) That the dog is not ill.
(*b*) That it is not fed.
(*c*) That it is not doped.
(*d*) That it trots sound.
(*e*) That its physical condition and the appearance of its skin are such that one would be prepared to support its inclusion in any programme.

Bearing this in mind, then, the veterinary surgeon should stop the dog as it comes up, have a quick look at its eyes for accommodation, smell its breath for recent alcoholic stimulants, watch its demeanour, and observe its abdominal contour. It should then be trotted up about 10 yards, turned sharply to have its balance tested and trotted back again.

Should the veterinary surgeon be satisfied, the dog should be permanently kennelled, but if there is a doubt, it is advisable to put the dog temporarily on one side or into its kennel from which later it can be taken and more carefully examined when the bulk of the kennelling is over.

Since the trainer will be held responsible for the running of the dog, the writer invariably asks his opinion should the demeanour or other feature call for query. Some greyhounds are normally sullen and others may be normally slightly pendulous in the abdominal region.

Since time is limited on all tracks, it is sound policy for the veterinary surgeon to watch the dogs as they move about prior to kennelling. It is often possible, during this period of ten minutes or so, to pick out dogs which do not appear to be normal, and this observation makes examination at kennelling time much easier.

Whole-time veterinary surgeons have usually examined runners at least once during the day of racing.

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Correspondence

Roaring in Cattle

Sir,—I was very much interested in the subject of "roaring" in cattle covered in the Questions and Answers section of the April 30th (1949) issue of *The Veterinary Record*. One of the things that aroused my curiosity was that no mention was made or suggestion offered in regard to the control of allergic symptoms by the use of antihistaminic or antiallergic compounds. I am not aware that cases of roaring in cattle, similar to those described by the enquirer in *The Veterinary Record*, occur with any degree of frequency in the United States or Canada, but one of my colleagues who operates a rather extensive animal practice (chiefly dairy cows) in New York State has been having excellent results by administering (intravenously) N,N-Dimethyl-N′-benzyl-N′-(alphapyridyl)-ethylenediamine or Pyribenzamine (Ciba) to cows suffering with oedema, urticaria and other symptoms that, judging by the quick response, are undoubtedly of an allergic nature.

Yours faithfully,
A. W. Rawson.

Ciba Pharmaceutical Products Incorporated,
Lafayette Park, Summit, New Jersey.
May 23rd, 1949.

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Angleberries in Cattle, Etc.

Sir,—In the U.S.A. good results are claimed, where these are not numerous, by the use of a wart vaccine. This is given subcutaneously, the dose being 25 c.c. injected at two or three sites and repeated once or twice at an interval of a week. Personally I prefer to adopt surgical removal, but it is of the utmost importance to take care that the instruments used are well sterilised afterwards, for I have known of direct transmission to another animal through failure to do this, *e.g.*, the use of scissors to snip these off a teat, and then their use to remove a supernumerary teat (an important preventative measure in any programme of mastitis control—quite apart from its blemish on a pedigree or prospective show animal) off another.

(May I for a moment digress and pass on a tip to my colleagues? The most satisfactory instrument to remove supernumerary teats is the Burdizzo emasculator. I use the smallest model, and where this fails to remove the pinched up skin and teat these are easily severed with a pair of straight scissors. Suturing can be left to the discretion of the operator. A dusting powder, preferably bismuth subgallate, is then applied.)

When an owner objects to surgical removal of these warts, oleate of arsenic can be applied twice a week.

Whilst yarding is commonly blamed for their occurrence, they are by no means confined to yarded cattle. The feeding of barley straw has been blamed for actinomycosis, but only once have I been able to incriminate it directly. Shortly before his death, Sir John M'Fadyean wrote asking me if I had been able to satisfy myself about this, as apparently he was rather sceptical. I replied as above, adding that I had encountered cases in cattle where gorse was abundant.

Yours faithfully,
J. F. D. Tutt.

"Rothiemurchus,"
St. Cross, Winchester.
May 26th, 1949.

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(*Continued on col. 1, page 392.*)

XIVth INTERNATIONAL VETERINARY CONGRESS

London, August 8th to 13th, 1949

TIME TABLE OF MEETINGS

A.—CENTRAL HALL. B.—HOARE MEMORIAL HALL. C.—WESTMINSTER SCHOOL.
D.—BISHOP PARTRIDGE HALL. E.—CONVOCATION HALL
HALLS B, D. & E. ARE SITUATED IN CHURCH HOUSE

Sunday, August 7th	Monday, August 8th	Tuesday, August 9th	Wednesday, August 10th	Thursday, August 11th	Friday, August 12th	Saturday, August 13th
	10 a.m. A. Official opening of the Congress.	10 a.m. A. The Veterinary Profession's contribution to the World's Milk supply Professor H. C. Bendixen	10 a.m. A. The Veterinary Profession's contribution to the World's Meat supply Dr. C.S.M. Hopkirk	10 a.m. A. The Veterinary Profession's contribution to the World's Poultry and Egg Supplies Dr. J. R. Beach	9.45 a.m. A. The Veterinarian and the Breeding and Rearing of Animals Professor N. Lagerlof	10 a.m. A. Closing meeting of International Congress
	11.15 a.m. A. The World Food Situation Lord Boyd Orr	11.15 a.m. A. Films	11.15 a.m. A. Films	11.15 a.m. A. Films	11 a.m. A. Veterinary Education and its Application to World Problems Dr W. R. Wooldridge	10 a.m. A, B, & D. National Veterinary Medical Association meetings continuing throughout the day
					12.15 p.m. A. National Veterinary Medical Association Opening Meeting	
2 p.m. to 2.30 p.m. A. Registration	2.15 p.m. A. 3 (*a*) Foot-and-mouth disease vaccination B. 3 (*e*) Swine fever	2.15 p.m. A. 2 (*c*) Bovine tuberculosis control B. 3 (*b*) Equine infectious anaemia	2.15 p.m. A. 2 (*a*) Bovine mastitis B. 3 (*d*) Newcastle disease	2.15 p.m. A. 2 (*e*) Salmonella B. 3 (*c*) Teschen disease	2.15 p.m. B. & D. National Veterinary Medical Association	
2.30 p.m. Permanent Commission meets in the Council Chamber in Church House	C. 6 (*a*) Surgery on the farm D. 4 (*a*) (i) Metabolism E. 5 (*a*) Plants as foodstuffs	C. 1 (*a*) Protozoal diseases D. 4 (*a*) (iii) Vitamins	C. 6 (*b*) Anaesthesia D. 4 (*c*) Infertility	C. 7 (*b*) Quarantine D. 5 (*c*) Genetics		
	3.45 p.m. A. 3 (*f*) Rinderpest	3.45 p.m. A. 2 (*d*) Tuberculosis	3.45 p.m. A. 2 (*b*) Brucellosis	3.45 p.m. A. 7 (*a*) Animal Products	3 p.m. Permanent Commission	
4 p.m. to 6 p.m. Tea and Conversazione at Ashburnham House	B. 3 (i) Swine influenza C. 1 (*b*) Helminth diseases D. 4 (*a*) (ii) Copper deficiencies E. 5 (*b*) Husbandry problems	B. 3 (*j*) Other virus diseases of cattle & horses C. 1 (*c*) Arthropod diseases D. 4 (*b*) Acetonaemia	B. 3 (*g*) Coryza C. 6 (*c*) Electrocardiography D. 4 (*e*) Endocrines	B. 3 (*h*) Rabies C. 8 (*a*) Veterinary Education D. 5 (*d*) Artificial insemination	3. 45 p.m. B. & D. National Medical Association	
6.30 p.m. Service, Westminster Abbey	Reception given by the President and Council of the Royal College of Veterinary Surgeons (Evening dress optional)	Banquet to Government Delegates given by His Majesty's Government (Dress : Lounge suits)	7.30 p.m. for 8 p.m. Congress Banquet, Savoy Hotel	6 p.m. to 7.30 p.m. Reception to all overseas visitors from the British Commonwealth, and their Ladies*	8.30 p.m. Reception given by the President of the National Veterinary Medical Association †	* Given by the Overseas League at Overseas House, St. James's, S.W.1 † By Invitation Dorchester Hotel, (Evening dress optional)

Recent Epidemic in Pigs in Ireland

Sir,—In your issue of May 21st you refer to a recent epidemic in pigs in Ireland.

This disease is not a recent epidemic. It has existed in this area for the past ten years, from about the time Lamont referred to it briefly as a disease of unknown origin.

The symptoms are as follows. Partial or complete paralysis of fore or hindquarters: the animal may drag the hind legs and have control of the fore, or the converse is often the case; frequently it walks on its knees and falls forward. Often the animal is on its side and uses all its limbs but cannot move from that position.

Oedema of the eyelids is common but not constant. Oedema of the pharyngeal region is frequent.

On *post-mortem* examination there may be no lesions. Oedema of the stomach, and also of the mesentery, however, is commonly seen. In cases I examined, the stomach was always distended with food, due, I believe, to paralysis of that organ.

Pigs of any age may be affected. In a lot of over sixty which consisted of three sows with litters varying in age from six to ten weeks and store pigs between three and four months, eight were dead and about 20 showed symptoms. One sow was dead and two showed degrees of paralysis.

The disease is commonest in young pigs from six to ten weeks. It may be very acute and animals die in a few hours. In mild cases the great majority recover. The disease makes its appearance suddenly.

I cannot understand why it is called "oedema disease" when the cardinal symptoms are those of a poliomyelitis and the condition is probably akin to the anterior poliomyelitis of the human.

As I have stated, this disease may be mild and a dose of laxative is helpful, but probably these would recover without any treatment.

Many years ago—I think in 1939—I wrote urging the responsible authorities to have the condition investigated, receiving the reply that it was a condition probably due to some deficiency in the food or to a vitamin want.

I hold—and will do so until the contrary is proved—that the disease is (1) a poliomyelitis; (2) caused by a virus; (3) airborne and probably transmitted also in other ways.

The financial losses sustained in one parish alone run into several thousands of pounds. It is difficult to make an approximately accurate estimate as the owner often conceals the condition. Apparently, from reports in the lay Press during the past year, it has spread all over the country and every class and condition of man has given an opinion on it.

Yours faithfully,

Bridge Street, New Ross, Co. Wexford. J. Lynch.
June 13th, 1949.

NOTES AND NEWS

Diary of Events

July 6th.—Summer Meeting of the Scottish Metropolitan Division, N.V.M.A. Excursion to A.I. Centre at Hoddom Castle, Ecclefechan, Dumfriesshire. (*See notice.*)

July 13th.—Meeting of the N.V.M.A. Editorial Executive Committee. 36, Gordon Square, W.C.1, 2.30 p.m.

July 15th.—Annual Meeting of the Association of Veterinary Teachers and Research Workers (Northern Region) at the Veterinary Field Station, "Leahurst," Neston, Wirral, 2.30 p.m.

July 15th.—Second Annual Meeting of the Association of Veterinary Teachers and Research Workers (Southern Region), at the Royal Veterinary College, Camden Town, N.W.1, 2 p.m.

July 15th.—Meeting of the Mid-West Division, N.V.M.A., at Bristol (Berkeley Café, Clifton), 2.15 p.m.

July 21st.—Summer Outing of the Central Veterinary Society to Newmarket. (*See notice.*)

July 22nd.—Meeting of the Royal Counties Division, N.V.M.A., at Oxford (Randolph Hotel), 12 noon.

Aug. 8th to 13th.—Fourteenth International Veterinary Congress, Central Hall, Westminster, and Church House, Westminster, London, S.W.1.

Aug. 12th to 15th.—Sixty-seventh Annual General Meeting and Congress, N.V.M.A., in London.

A Welcome "Reversion to Type"

In the typographical sense that means so much both to the appearance of the journal and the comfort of the reader, with this issue *The Veterinary Record* becomes itself again. We refer, of course, not to the product of pre-World War I, which was deficient in many respects, but to that featuring the 10 point-size of type-face introduced shortly before the war, when we adopted that also selected by *The Times* primarily on its optical merits. The benefit of the change was short-lived, disappearing with the advent of a decade during which, in the interests of space saving, the noble amplitude of the new type had to give way to the relative meanness of "8 point" and the latter (for all "tabular" and much other matter) to the paltriness of "6"—a type which threatens alike the eyes and temper of all condemned to read it and is wisely ignored by those under no obligation to do so. Thus with the use of larger type for all major features is coupled the boon of abolition, from both the *Record* and its Supplement, of all type of microscopic size.

In effecting these beneficial alterations at this half-year there is a symbolic timeliness. It is indeed fitting that a new liberality in mode of production of our periodical, limited only by financial considerations, should accompany the vast expansion in veterinary thought and activity epitomised in the great International Veterinary Congress and that of our own Association, opening in London at the beginning of next month.

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The "International"

In this issue publication is made of the time-table of events of the forthcoming XIVth International Veterinary Congress. The organising staff are working under many difficulties, therefore the co-operation of all those intending to be present at the Congress is earnestly requested. It will assist the staff greatly if intending members who have not yet enrolled will do so without delay.

The following amendments should be made to the provisional programme of papers printed in our issue of May 7th: Section 5 (b): Mr. W. G. Beaton comes from Nigeria and not Kenya as stated. Delete paper by Mr. Hay Barclay.

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PERSONAL

Appointment of Mr. W. L. Weipers as Director of Veterinary Education in the University of Glasgow

Glasgow University Court have appointed Mr. W. L. Weipers, M.R.C.V.S., to be Director of Veterinary Education in the University from October 1st, when the Glasgow Veterinary College will be incorporated into the University. Until new buildings can be erected the work of the College will be continued at Garnethill. Further reference will be made in our next issue.

Appointments.—Mr. J. J. Thompson, M.R.C.V.S., has resigned his position as Assistant V.I.O. with the North of Scotland College of Agriculture, Aberdeen—a post which he has occupied since 1945—to take up the appointment of Poultry Veterinary Officer, Professional Division, New Zealand Department of Agriculture.

Mr. Thompson left this country on July 1st, and prior to his departure from Aberdeen he was the recipient, as he writes, "of a very fine wrist watch from the members of the North of Scotland Division, N.V.M.A., of which I was Hon. Treasurer since 1946. Unfortunately (he continues) I did not have the watch in time to thank the members of the Division at a recent meeting, but I should appreciate it very much if I were able to express my thanks to them through the courtesy of *The Veterinary Record*."

Mr. Stuart Young has been appointed Assistant V.I.O. with headquarters at Mill of Craibstone, and Miss Margaret Cooper, Assistant V.I.O. with headquarters at the sub-laboratory at Glensaugh Hill Farm Experimental Station.

Births.—Greenough.—On May 31st, 1949, to Pauline, wife of Paul R. Greenough, M.R.C.V.S., of Church Cottage, Mersham, Nr. Ashford, a son—Timothy John.

KAY.—On June 9th, 1949, at Rotherham, to Mary (*née* Brown, L.D.S.), wife of Robert S. Kay, B.V.SC., M.R.C.V.S., a son.

WILKINSON.—On June 25th, 1949, at Elmswood Nursing Home, Liverpool, to Susan (*née* Smith), wife of George T. Wilkinson, B.V.SC., M.R.C.V.S., a daughter—Linda Susan.

WOOD.—On June 24th, 1949, at the Royal Cornwall Infirmary, Truro, to Barbara, wife of Douglas R. Wood, Ashby Villa, Tregoney, Truro, a daughter—Judith Anne.

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The Late Mr. Thos. Wilkinson, M.R.C.V.S.

In our issue of June 18th we recorded, with deep regret, the death of Mr. Thomas Wilkinson, M.R.C.V.S., of Lanchester, who was a beloved figure in North-West Durham, and one actively engaged in the agricultural life of the community.

Born in Lanchester, he qualified in 1897, at the Edinburgh Veterinary College and served five years with Messrs. Bolckow and Vaughan at Eston. He returned home to assist his father and worked throughout the area until he retired from active practice in 1934.

For many years Mr. Wilkinson farmed Moor Leazes in the village and represented the farming community on many organisations, including the N.F.U. and the County War Agricultural Committee. He was Chairman of Lanchester branch N.F.U. and President of Butsfield Young Farmers' Club from its inception. President of the North of England Veterinary Medical Association, he was also a member of Council, N.V.M.A., for a long period. A Freemason, Mr. Wilkinson held high office in the Craft.

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Cambridge School of Veterinary Medicine

For the first time Cambridge is to embark on veterinary teaching. The University has agreed to seek powers under the Veterinary Surgeons Act, 1948, to confer a degree in Veterinary Medicine that will be a registrable qualification, and in October a limited number of veterinary students will begin a six-year course leading to this degree. The first three years will be spent in study for the Natural Sciences Tripos, and the following three years in professional studies. Plans are being drawn up for the erection of buildings for the new School of Veterinary Medicine, which will be situated within two miles of the centre of Cambridge on an area of approximately 120 acres of farmland. As with all courses in Cambridge, candidates must first be accepted for admission by a College, and it is unlikely any College will accept for admission as a veterinary student an applicant who has not passed three of the four parts of the first M.B. Examination.

Financial provision for these courses will be made by the University Grants Committee, which has agreed to provide a grant of £31,000 for 1949-50.

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R.C.V.S. Council Election Statistics

We publish herewith certain figures regarding the last four elections to Council, R.C.V.S. It is interesting to note that there was a considerable increase in the number of voting papers sent in for the recent election over the previous three years, whilst on the other hand the percentage of votes cast by each voter remains reasonably constant.

Year	*Candidates for 8 places*	*Voting Papers*	*Votes Cast*	*Number of Votes used by each voter out of a total of 8 possible*
1946	14	1,215	8,304	6·8
1947	9	912	6,210	6·8
1948	11	1,113	6,570	5·9
Year	*Candidates for 20 places*	*Voting Papers*	*Votes Cast*	*Number of Votes used by each voter out of a total of 20 possible*
1949	25	1,466	24,326	16·5

In connection with the increased poll this year it should be noted that for the first time the ballot paper sent to voters overseas was identical with that sent to voters in Great Britain and Northern Ireland. This was due to the fact that an early date was made for sending voting papers out, whether at home or overseas. It is understood that as a result of this there was a considerably increased overseas poll.

Next Year's Election

The election next year will be for five members only, as five elected members will retire in accordance with the provisions of the Veterinary Surgeons Act, 1948, unless of course there are any vacancies to be filled over and above the five retiring members.

In the autumn we shall publish, as we did in the autumn of last year, an article giving full instructions regarding the procedure to be adopted for the election in 1950.

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ROYAL ARMY VETERINARY CORPS

Officers' Club Annual Dinner

The Annual Dinner of the Royal Army Veterinary Corps Officers' Club was held at Grosvenor House on Friday, June 10th. General Sir Sidney C. Kirkman, Quartermaster-General to the Forces, was the guest of the evening and Brigadier J. J. Plunkett, Director, Army Veterinary and Remount Services, presided.

The following were present:—

Brigadier J. J. Plunkett, C.B.E.

General Sir Sidney C. Kirkman, K.C.B., K.B.E., M.C.

Major-General J. J. B. Tapley, C.B., D.S.O.

Brigadiers G. A. Kelly, C.B.; H. S. Mosley, D.S.O.; C. A. Murray, C.B.E.; E. S. W. Peatt, O.B.E.; J. J. Kane, O.B.E.

Colonels R. M. Bamford, O.B.E.; G. Barnett, O.B.E., M.C.; H. E. Gibbs, D.S.O.; C. W. Heane; J. J. Mills; W. L. Sinton, M.B.E.; A. G. Todd, C.B.E., D.S.O.; C. H. S. Townsend, C.B.E., M.C.; W. H. Walker, D.S.O., O.B.E.; P. F. Woodland.

Lieut.-Colonels V. A. Bartrum, O.B.E.; J. Bell; J. C. Bennison; J. Clabby, M.B.E.; R. C. Crowhurst, O.B.E.; F. L. Cundell; B. C. M. Edmonds; C. P. Fisher; O. S. Fisher, O.B.E.; D. A. Green; J. Hickman; L. H. B. Poer, M.C.; J. McL. Ross, M.B.E.; W. M. Rouse,; D. F. G. Smith; D. I. C. Tennant; G. F. Watkins; H. B. Williams, O.B.E.

Majors E. Brayley-Reynolds, O.B.E.; M. Bridgeman, T.D.; D. C. E. Danby; C. Davenport, O.B.E.; P. W. Dean; J. J. Dunlop, M.C.; A. V. Franklin, T.D.; J. Going; E. D. Greenway; J. Harrison; C. H. H. Jolliffe; T. Lishman; R. I. Macrae; H. P. J. Murray; G. T. Newport, M.B.E.; A. D. Seton; W. E. Turton; E. Wallace; W. J. B. Watson; G. D. Young.

Captains R. H. M. Coulton; W. Harris; P. J. Mitchell; J. H. Wilkins.

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LIVESTOCK EXPORT GROUP

Mr. A. E. Baldwin, M.C., M.P., and Mr. F. Nevill Matthews, M.B.E., have been appointed Chairman and Vice-Chairman, respectively, of the Group. The names of members comprising the new General Committee are as follows:—

Three members appointed by N.C.B.A.—Mr. W. J. Cumber, Mr. J. H. Everall and Brigadier E. R. Kewley.

Two members appointed by N.P.B.A.—Mr. A. R. Bennett and Mr. J. T. Eady.

One member appointed by Essex Pig Society—Mr. J. G. Valentine.

Three members appointed by N.S.B.A.—Major T. K. Jeans, Mr. A. N. Bocock and Mr. T. C. Norris.

Three members appointed by Thoroughbred Breeders' Association—Lord Rosebery, Mr. F. E. Birch and Mr. Adrian Scrope.

One member appointed by British Horse Society—Brigadier W. H. Anderson.

One member appointed by British Goat Society—Mr. T. W. Palmer.

One member appointed to represent Poultry—Miss E. E. Kidd.

Members appointed to represent Shippers and Exporters—Mr. J. A. Arnold, Mr. J. Biggar, Mr. R. P. Evans, Colonel D. Kennedy, Mr. W. Lloyd, Mr. A. J. Marshall, Mr. H. Nickson, Brigadier R. S. Scott and Mr. R. R. Timlett.

One delegate appointed by R.A.S.E.—Lord de La Warr.

One delegate appointed by N.V.M.A.—Dr. R. F. Montgomerie.

One delegate appointed by Board of Trade—Mr. A. Sherrard.

Two delegates appointed by Ministry of Agriculture and Fisheries—Mr. A. M. Graham and Mr. R. Harding Wynne.

One delegate appointed by Dept. of Agriculture for Scotland—Mr. Wm. Ness.

One delegate appointed by Royal Highland and Agricultural Society—Mr. W. Hogg.

The following members have been elected by the General Committee to form the Executive:—

Seven members representing Breeders' interests.—Lord Rosebery (Thoroughbred horses), Dr. R. F. Montgomerie (dairy cattle), Brigadier E. R. Kewley (beef cattle), Mr. A. N. Bocock (sheep), Mr. J. T. Eady (pigs), Brigadier W. H. Anderson (horses) and Miss E. E. Kidd (poultry).

Five members representing Shippers and Exporters.—Mr. J. Biggar, Mr. R. P. Evans, Colonel D. Kennedy, Mr. H. Nickson and Brigadier R. S. Scott.

Lord Lovat, Mr. J. L. Cridlan and Mr. John Craig were co-opted to serve on both Committees.

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the N.V.M.A.

Correspondents are requested to write as briefly as possible.

DEMODECTIC MANGE

Sir,—The more I contemplate this disease of the dog, the more I become convinced that it is not only congenital but that puppies may be born already infected. Numerous instances have come to my notice over a number of years which should have led me earlier to this conclusion, but unfortunately I have not in the past been sufficiently methodical to make detailed records of the case histories and am, therefore, now somewhat at a loss to supply sufficient of the proof which readers no doubt would demand.

The reasons which have prompted me to make this statement are, in the main, five, *viz.*:—

1. Numerous clinicians and others have tried deliberately to bring about infection of healthy dogs by causing them to live and to sleep in the same box with affected ones. I think I am correct in believing that the almost—if not entirely—universal verdict has been that contagion did not take place.

In fairness, however, I am bound to record one contradictory case, and the only one in my recollection, in which a ten-year-old Dachshund had developed the disease, although, according to the owner, neither its parents nor grandparents had ever shown lesions.

It is possible, of course, that parents may actually harbour the parasite and impart it to the foetus and yet never exhibit noticeable symptoms of its presence.

2. The second reason is the fact that this acarus has been demonstrated in faeces, lymphatic glands and blood. It would appear, therefore, that the mite may remain latent in the blood stream or may one day make its way out of the superficial capillaries and become deposited in the hair follicles. We have yet to know what lapse of time or what conditions must pertain to allow this change of nidus. We do know that puppies may exhibit lesions from about two weeks to several months of age, and that in some cases they have actually shown lesions at birth. In support of the latter statement I quote an extract from a client's letter (name and address of the client are available to any person interested): "Some miniature Dachshunds came to me a year ago and one of them was continually shaking but did not show outward signs of follicular mange until she had puppies *which were born with it*, and then *she* showed signs of it."

If the blood of an affected dam harbours the parasite then, of course, this circulates in the foetus and the puppy has been infected pre-natally. What is it that determines whether lesions shall not be visible for several weeks, months or years after birth or may never appear at all?

3. Thirdly, the fact that such a large proportion of cases of demodectic mange occur in Dachshunds *does* strongly suggest that the disease is passed on from parents to offspring time and time again. It is thus not a condition which is passed from skin to skin, but which is inborn or congenital, in much the same way as we find night-blindness is transmitted in Red Setters and entropion in Chows.

4. Fourthly, the case which occurred in Frank Chambers's practice and which was illustrated and recorded in my "Index of Diagnosis." This concerned the affection of two young Dalmatian puppies born of a bitch three successive litters of which had all developed this disease at about four months of age. The bitch had never shown any lesions at any time.

Furthermore, other entire litters have been known to develop the disease even though all members of the litters had been separated from the dam and from each other, the dam having shown no symptoms.

5. Lastly, there is great significance in the fact that quite recently reports have come from Africa that phenamidine, introduced by subcutaneous injection, has cured a number of cases and in the absence of any local application whatever. Phenamidine is a drug which was primarily directed against Babesiosis and it was accidently discovered that whilst being subjected to this treatment dogs simultaneously affected with the Demodex became cured. There is little reason to doubt the veracity of these reports, but if they are true then we find that demodectic mange is being successfully combated from within and that treatment of the skin itself is of secondary importance. This drug was brought to my notice only 2½ months ago, since when only two cases of demodectic mange have been presented to me—both in Dachshunds.

The first was a five-month-old puppy which, in appearance, was absolutely grotesque. Its skin was so corrugated and striated that it resembled a zebra. Six injections of phenamidine were given at three- to four-day intervals in a dosage of 0·3 c.c. per kg. Ten minutes after each injection the dog would show great irritation, shaking its head and evincing general agitation. This would pass off within half an hour. At the end of a month the animal was practically cured and all striation gone. Just then the second case came in and the same treatment was started. Eight injections in this case failed to achieve anything except emesis and accelerated respiration after each injection. Further opportunities for testing out the method are awaited.

So far as topical treatment is concerned, it seems to me that many agents can be relied upon to kill the superficial acari, but what troubles me is to know whether a demodex—transferred from the skin to a slide—is dead or alive. They appear never to move at any time one may observe them. Can it be that they shrink and gradually dissolve when killed?*

It may be conceded that many agents such as Lorexane, balsam of Feru, organic sulphurs, rotenone, etc., etc., are lethal to those that can be reached by topical application, but how are we to prevent the continuous reinvasion of the follicles from within?

I do believe that our only hope of overcoming the disease is through the agency of a drug parenterally introduced, and phenamidine may prove to be the drug.

Yours faithfully,

658, Finchley Road, N.W.11. HAMILTON KIRK.

* I cannot believe this can be true because I have kept specimens on slides for many months and under the microscope their appearance (after all that time) has in no way differed from that when they were first detected.—H. K.

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SOME CLINICAL OBSERVATIONS ON MILK FEVER

Sir, When I qualified I took over the premises and equipment of the late Mr. Dobbyn, of Waterford. He had been accustomed to treating milk fever cases by the inflation method, using oxygen, a method I continued to use with success. However, after some time, in cases of relapse which had been inflated previously either with air or oxygen, I noticed that if I attempted re-inflation with oxygen the animal died before the process was completed. Writing from memory, I think I had five such cases. I could not account for the cause of death, which, however, seemed to be of the nature of anaphylactic shock. I then commenced to inflate such relapsed cases with air, with the result that these casualties ceased.

I have always noticed that one case of milk fever invariably is followed by another on the same day—in fact I have had as many as five in one day—and probably none for at least a week afterwards; this inclines me to think that perhaps the weather has something to do with the onset of the condition: on hot sultry days I notice that milk fever is more prevalent.

As previous writers have recorded, I find that one has occasionally to resort to inflation where borogluconate has failed. However, I would like to record a typical case of milk fever which was staggering round the byre whilst I was injecting her subcutaneously with 3 oz. of borogluconate. I assured the owner that the animal would be normal in a few hours, but 12 hours later I received an urgent message to go again. I found the cow completely prostrate and comatose, and was informed that she had got steadily worse from the time of injection. I decided to inflate her with air and in 30 minutes she was able to sit up, but after 12 hours there was no further improvement and the animal would take nothing to eat. I injected 3 oz. calcium borogluconate and in another 30 minutes she had commenced to eat; from that time on she made an uneventful recovery.

I would be glad if I could obtain some explanations of the above phenomena.

Yours faithfully,

Charlemont Cottage, Moy, Co. Tyrone. GEORGE HOBSON.

June 16th, 1949.

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R.C.V.S. COUNCIL ELECTION

Sir,—May I avail myself of the opportunity of thanking the electors who voted for me at the recent Council election.

I can assure them that it will be my earnest endeavour, as in the past six years, to give freely of my services in the important work of the Royal College and uphold the dignity of our profession.

Yours faithfully,

20, Ramsden Street, Huddersfield. W. R. MCKINNA.

June, 1949.